THE RELATIONSHIPS AMONG LEISURE TIME ACTIVITIES, MAXIMUM OXYGEN CONSUMPTION, LIPID LEVELS AND PERCENTAGE BODY FAT IN A GROUP OF SEDENTARY OFFICE WORKERS

by

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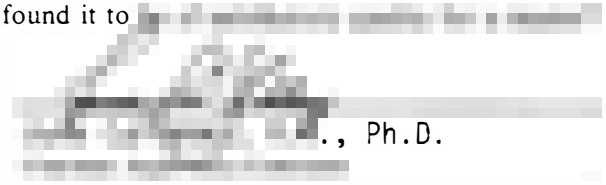

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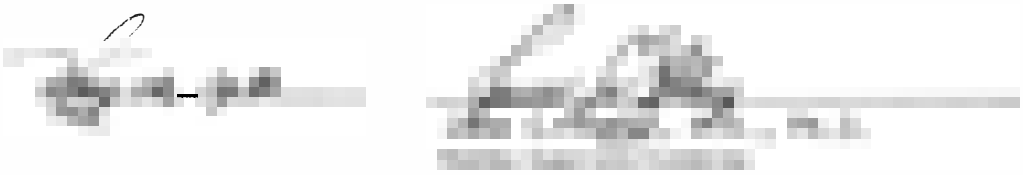

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ABSTRACT

Relationships between leisure-time physical activity, maximum oxygen consumption (MVO_2) lipid levels, blood pressure, and percent body fat were investigated in 50 sedentary male office workers. Physical fitness, as evaluated by the Fisher-Fairbanks Walking Test showed a small, but statistically significant positive correlation with High Density Lipoprotein (HDL) cholesterol ($\underline{r}$ = .33). Total Activity Metabolic Index also demonstrated a small but statistically significant correlation with HDL cholesterol ($\underline{r}$ = .20). Body composition may reflect to an extent, level of regular physical activity or fitness, since caloric expenditure is a determinant of fat deposition in or removal from adipose tissue. Maximum VO_2 showed a negative correlation with percent body fat ($\underline{r}$ = -.35). In the multivariate analysis, knowledge of cholesterol, VLDL, diastolic pressure of total Activity Metabolic Index (AMI) added no prediction beyond VO_2, HDL and VLDL ($\underline{r}$ = 0.85) for determining placement in physical fitness categories.

In loving memory of my father, Kenneth Louis Quaal.

Education should teach how to think, not what to think.

John Dewey

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

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CHAPTER I

INTRODUCTION

The killing diseases of a generation ago were largely caused by infectious agents. In more advanced communities the spread of these diseases has been controlled by public health measures or preventative immunization. Bacterial infections, today however, strike down mostly only those enfeebled by age or ill health arising from other causes, but we fear the "metabolic" disorders. We may not know in a given case if the cause is an environmental factor or an apparently spontaneous metabolic derangement, of perhaps genetic origin. The disentanglement of the underlying factors is complex. It is to biochemistry that we must look for preventative or remedial measures, just as a generation ago we looked to bacteriology.

Most attention has been focused on coronary artery disease (CAD), specifically lipid infiltration of the intima layer of arterial vessels, since this is the phenomena which presents itself to the medical practitioner and to the public registrar of deaths. The dramatic increase of this disease during the past quarter century is causing great concern. Autopsy studies have shown that young men and women, even teenagers, have the beginning of atherosclerosis. Coronary artery disease attracts public attention because the disease claims most victims amongst leaders of the societies in which it is prevalent. Among the male members of the executive and professional

classes in the U.S.A., CAD is the single most important cause of death. In 1977, 984,972 people died from heart and blood disease--52% of deaths from all concerns. Over 40,000 Americans have some form of heart and blood vessel disease. Heart and blood vessel diseases will cost the nation an estimated $40.8 million in 1980. Other "hidden" costs--losses in management skills, production "knowledge" personnel training and development and labor turnover, are difficult to estimate (Heart Facts, AHA, 1980).

In the introduction of Eliot's volume, Stress and the Heart (1974), the late Paul Dudley White noted important changes in the symptomatic manifestations and frequency of occurrence of cardiovascular disease during the course of his highly productive career. In the forward to this same book, Robert S. Eliot suggests that human distress and the incidence of coronary death have shown parallel increases during the 20th Century.

During the past half-century, credible clinical and pathologic observers have measured the immense increase of myocardial infarction and sudden death. Multimillion dollar programs, however, have failed to clearly identify the required cause and effect data. Atherosclerosis remains a mystery. The time-honored sine qua non of "heart attack," indeed infarction, can be logged without any coronary luminal narrowing.

Since both human stress and the incidence of cardiac death have increased in parallel during this century, interrelationships have been sought between the two. Selye (1950) and Raab (1970), among others, have led the way toward clarifying the role of adrenal

cortical mechanisms interrelating stress to myocardial oxygenation. Much remains to be done in sorting out the complex cooperative mosaic of the factors influencing myocardial oxygenation. Fundamental to our control of premature cardiac death, however, is this type of understanding, which must balance the plumbing aspect of the coronary arterial system with myocardial factors, blood oxygen transport capability and metabolic interactions.

Society of late appears to reward a man in a fashion and to the degree that he functions as a robot chugging obediently toward ambivalent and often artificial goals. The submerging of human identity has tended to reduce the capability for appropriate human response. Dissatisfaction has emerged with the recognition of empty or inappropriate rewards. When complaints have fallen on deaf ears, there has developed anxiety which has become translated into further dissatisfaction, both individual and collective.

Material rewards do exist, but are often empty in meaning and subject to mutual unrest and discontent. For example, for those seeking travel as a diversion or as a business necessity, air traffic controller-shutdowns can both bind and imperil the traveler into a stone age level. The automobile has sunk from a status symbol to a four-wheeled example of the ecologic and energy crisis. Thus, the dissatisfaction engendered by inadequate and inappropriate reward has contributed to the development of an era of human stress, precipitating coronary disease.

The swing towards a coronary break down is rarely spontaneous. The difficulty in finding and dealing with the cause lies in the

human tendency to deny its existence. Work is very deeply involved in these individuals' sense of duty and conscience. Very striking is the tendency to accept challenges and to assume more tasks than can leisurely be fulfilled. Work seems to be the main way in which they strive to climb the social scale.

Researchers have suggested that excessive work and responsibility, when approaching the limits of the individual capacity to control the work, precipitates the biochemical changes which accelerate coronary artery disease (Nixon, 1976). Executive health is of interest when one searches for a population which may, unknowingly exhibit these physiological manifestations of impending coronary breakdown. Many individuals exhibiting biochemical changes which make them more susceptible to CAD are completely unaware of the physiological manifestations they are exhibiting, or they may make strong declarations of health and virility that are at odds with the observed behavior. Excessive burdens and pressures, disruptive of health and happiness are accepted as inevitable because the exhaustion reduces the ability to distinguish the essential from the nonessential.

To be set up in healthy function implies that healthy human function can be achieved and maintained if the individual respects the inexplorable laws of nature and chooses not to force himself into the over-aroused and exhausted life styles which generate self-defeating changes in blood chemistry and blood pressure. Nixon (1976) has found that one of the best ways of achieving a greater workload without succumbing to arousal and exhaustion is to train to become fit enough for the successes desired. The untrained individual

who becomes aroused when subjected to exhaustive and provocative circumstances needs to be made aware of the fact that he is at greater risk for the development of CAD.

An objective indicator of the functioning of the cardiovascular system now being incorporated into medical examinations is the response of the heart to muscular work demands. The mortality from heart disease in Swedish males 35 to 44 years of age, is about one quarter that of their American counterpart (Cooper, 1969). The fact that Swedes have a higher maximal oxygen consumption (MVO_2) level at younger ages lends support to the contention that a higher capacity for exercise might be a reflection of cardiovascular health.

The hypothesis that habitual physical activity protects against coronary heart disease is derived mainly from studies which show a relationship between levels of exercise involved in different occupations and the incidence, prevalence and mortality rates of this disease. One must consider, however, the level of physical activity during leisure which is not accounted for when occupational physical activity alone is studied. If we are to make use of exercise in preventing coronary heart disease, we must concentrate on leisure time exertion. With the increasing mechanization of jobs, fewer and fewer occupations involve significant physical exertion, and the study of these occupations is less relevant to the main problem of coronary disease. Therefore the scope of this experiment will center around investigation into the relationships between the amount of physical activity engaged in outside of work and max VO_2, lipid levels, percent body fat, weight, and blood pressure.

Statement of the Problem

Although a review of the literature reveals lowering of the lipid profile with marathon running and temporary brief periods of normalization in plasma lipid levels in hyperlipidemic men following a single period of work, no study has been undertaken to compare lipid profiles, physical fitness and percent body fat composition among a homogeneous group of office workers with respect to leisure time activities. Therefore, it appears timely to investigate the relationship of these variables. The problem can be stated as thus: Among a group of homogeneous sedentary office workers, aged 30-55, without clinical manifestations of atherosclerosis, will there be demonstrated a correlation between leisure time physical activity aerobic fitness, plasma lipid profiles, body fat and blood pressure?

Hypotheses

1. There will be a positive correlation between Total Activity Metabolic Index (AMI) and High Density Lipoprotein (HDL), and a negative correlation between AMI and cholesterol, Low Density Lipoprotein (VLDL), Low Density Lipoprotein (LDL), and triglycerides.

2. There will be a positive correlation between percent body fat and cholesterol, LDL, VLDL, and triglycerides and a negative correlation between percent body and HDL.

3. Subjects with higher Total AMI scores will have higher VO_2 values.

4. Subjects with higher VO_2 scores will demonstrate higher HDL values.

5. Subjects with higher VO_2 scores will demonstrate lower LDL.

6. Those subjects with higher VO_2 scores will demonstrate lower triglycerides and cholesterol.

Assumptions

It was assumed that:

1. Each individual reported with reasonable accuracy his leisure time activities.

2. From the time that the subjects agreed to participate in the study to the time of data collection (2-3 weeks) there was not a marked change in leisure time activities.

3. Each individual answered the questions truthfully in the screening questionnaire for evidence of coronary disease, hypertension, hyperlipidemia, pulmonary disease, infections or physical activity restrictions.

CHAPTER II

REVIEW OF THE LITERATURE

In considering a separation of areas for efficiency in reviewing the related research, it becomes apparent that the parameters of this experiment are invariably interrelated and interwoven throughout most of the available research. The purposes of reviewing the related research are to provide insights into procedures used in other experiments similar to the present experiment, to provide useful information, to provide a sample of various facts and to provide data for comparative purposes.

Lipids and CAD

Serum cholesterol, triglycerides, HDL, VLDL, LDL have all gained notoriety in association with coronary heart disease. On a clinical level, Friedman, 1970, has demonstrated that the lipid regulating mechanisms are responsive to stressful life experiences and that elevations of serum cholesterols, triglycerides, blood sugar and uric acid are associated with a greater incidence of clinically manifested coronary artery disease. Elevated levels of one or more lipoprotein classes are common in the general population. They have been identified by Fredrickson by the use of the Roman numerals I through V (Fredrickson, 1975). Of these five types, only two are commonly encountered: Type II characterized by excessive concentrations of LDL and Type IV hyperlipidemia characterized by excessive

concentrations of VLDL.

Recently serum cholesterol has gained notoriety as being more closely associated with the cause of coronary heart disease than any of the other generally accepted coronary risk factors. As a result, an overwhelming amount of research has been completed on the subject of what affects the serum cholesterol level in humans. This section presents a sampling of some of the more popular views.

Kannel, et al., 1971, have been conducting the Framingham Study continuously since 1949. At the time of the initial examination in Framingham, 2,282 men and 2,845 women aged 30 to 62 were examined and found free from coronary heart disease. Examinations on a variety of serum lipids, among them cholesterol, were conducted on the 5,127 subjects biennially. The subjects were then classified into groups according to the first two biennial examinations. The purpose of the Framingham Study was to determine risk factors related to coronary heart disease and establish priorities to these risk factors. Of the original study group, free from coronary heart disease, it was possible to re-examine 80% on the eighth biennial examination, representing 14 years of follow-up.

In the 14 years of follow-up 323 men and 169 women between the ages of 32 and 62 years at initial examination developed for the first time some clinical manifestation of coronary heart disease. (The diagnostic criteria used to detect coronary heart disease were the clinical manifestations of angina pectoris, myocardial infarction, the coronary insufficiency syndrome, and sudden unexpected death.) The mean level of each of the major serum lipids was higher, at the

initial examination, in those who went on to develop coronary heart disease than in their cohorts, who remained free of clinical manifestations of the disease over the 14 year period of observation. Re-examination of the relation of the level of each of the major blood lipids under consideration after 14 years of observation continued to show a distinct and striking increment in the risk proportional to the lipid concentration. The relationship was generally stronger in younger than older persons.

As a determinant of risk, none of the lipids or lipoproteins measured appeared to be superior to serum cholesterol. The mean cholesterol value for the 5,127 subjects was slightly below 220 mg./100 ml. of blood. Moderate serum cholesterol elevations between 250 and 350 mg./100 ml. constitute the bulk of the "hypercholesterolemias" that appear to be predisposing to the abundance of coronary heart disease as it occurs in the general population. Such levels constituted the upper quartile of the Framingham Study. It is interesting to note that six individuals were found to have initial cholesterol levels greater than 400 mg./100 ml. and a strong family history of coronary heart disease and within the follow-up period all six died of coronary heart disease before their fiftieth birthdays (Kannel, 1971).

S. R. Shane reviewed the records of 877 flying personnel evaluated at the School of Aerospace Medicine during 1963 and 1964, who had undergone treadmill exercise testing and determination of their serum lipids (1966). In this study an attempt was made to correlate serum cholesterol with different levels of physical

conditioning. Physical condition was established utilizing the Balke-Ware Treadmill Test. When the subject's heart rate reached 180 beats per minute, the expired respiratory gases were collected for one minute to determine the maximal oxygen uptake of each subject.

The results of the study indicated a significant inverse correlation involving serum cholesterol and either time on the treadmill or maximal oxygen uptake, both of which can be used as measures of physical condition. These data indicate that the degree of physical condition can influence serum cholesterol and that the better the physical condition the lower the serum cholesterol level is (Shane, 1966).

Fyfe (1968) made 3,701 estimations of serum cholesterol between January, 1965 and December, 1966. The overwhelming majority of the determinations were performed on patients with confirmed or suspected coronary heart disease. The authors plotted the serum cholesterol values on a graph by months of the year to determine whether or not a seasonal variation existed. The results indicated that a highly significant variation in serum cholesterol existed. The highest levels occurred in the spring and the lowest in the fall. When the cholesterol variations were correlated with the possibility of similar seasonal variations in the incidence and mortality of coronary heart disease, no correlations were found.

Garrett conducted a study of the effects of a strenuous physical conditioning program on coronary risk factors in men. Thirteen subjects were selected based on levels of cholesterol, blood pressure, and obesity. The study was divided into three

phases, a pre-training phase during which time baseline measurements were obtained for the variables to be tested, the training phase, and a post-training phase. The phases were four, six, and eight weeks in duration, respectively. Measurements were repeated at the end of the training phase and again at the end of the post-training phase. Garrett reports that the experimental group's mean body weight went down significantly as did the levels of serum cholesterol. Physical fitness levels increased significantly after the training phase. Diastolic blood pressure levels were significantly reduced at the end of the training phase but these levels returned to pre-training levels after the post-training phase. Systolic blood pressure levels were not significantly affected by the training phase (Garrett, 1965).

Campbell investigated the influences of several physical activities on serum cholesterol concentration in college freshmen. One hundred and thirty-three subjects were randomly assigned to the following activities: cross-country running, weight training, tumbling, wrestling, tennis, golf, and a control. Serum cholesterol levels were measured before and after training by a method prescribed by Abell. The mean initial serum cholesterol level for all the groups was 177.4 mg./100 ml. of blood, with the mean average change for all groups being a -1.94 mg./100 ml. of blood. However, it must be noted that these are means and that the individual groups did vary in amount of change. Campbell found that cross-country running produced a significant decrease in serum cholesterol when compared with all other activities except tennis. It was concluded that

cross-country running and tennis were phasic activities and produced decreases in serum cholesterol proportional to their relative intensities, whereas the more static activities of wrestling, weight training, and tumbling produced little or no detectable change (Campbell, 1966).

Campbell (1966) studied the influence of physical activity on blood serum cholesterol levels of young men. Volunteer subjects were tested by Behnke's method to determine where the subject was classified and weighed; they were randomly placed into active and inactive groups. Following this, the subjects were pretested for cholesterol levels. The initial mean serum cholesterol level for the active groups was 193.34 mg./ 100 ml. of blood, and the same level for the inactive groups was 195.15 mg./100 ml. of blood. The active group ran on a treadmill three times per week for ten weeks, whereas the inactive did not train. Posttests for cholesterol levels and body weight were given following the 10-week training period. The final mean serum cholesterol level for the active groups was 187.61 mg./100 ml. of blood. The active groups showed a mean change in serum cholesterol resulting in a decrease of 5.73 mg./100 ml. of blood, whereas the inactive group's mean change was a decrease of 1.85 mg./100 ml. of blood. There was a significant difference between active and inactive subjects and between groups in the levels of blood serum cholesterol, whereas the mean changes in body weight were not significant. The greatest mean reduction in serum cholesterol and body weight occurred in the obese active group.

Montoye (1966), studied the relationship between serum

cholesterol and body fatness. Tests were run on the majority of the population of Tecumseh, Michigan. Correlations were determined for all individuals taking the tests on serum cholesterol levels and body fatness. Measurements were taken using a skin-fold caliper and Abell's method for measuring cholesterol. A low (.43), but statistically significant, relationship was found between serum cholesterol levels and body fatness.

Montoye and colleagues used 31 faculty members at Michigan State University to investigate the effects of exercise on blood cholesterol. The subjects were pretested for initial levels of cholesterol and then randomly placed into two groups. Group I was the exercise group. Their initial mean serum cholesterol level was 189.5 mg./100 ml. of blood and their mean age was 43 years. Group II served as the control. Their initial mean serum cholesterol level was 194.6 mg./100 ml. of blood and their mean age was 41 years. Five subjects whose initial mean serum cholesterol level was 239.8 mg./100 ml. of blood were judged to be initially high level subjects and were treated separately. Three of these men exercised and the other two were controls. The exercise group underwent 3 months of supervised exercises, whereas the control group remained inactive. Upon completion of the training period the subjects were posttested. The exercise group's final mean serum cholesterol level was 187.1 mg./100 ml. of blood and the same level for the control group was 196.6 mg./100 ml. of blood. The initially high-level subjects' final mean was 212.4 mg./100 ml. of blood. These subjects also lost the most weight during the study. The results indicated that a change in total

serum cholesterol generally accompanied as a change in body weight. Exercise was therefore effective indirectly by decreasing the body weight (Montoye, 1949).

Campbell (1968) studied the effect of controlled running on serum cholesterol of young adult college men of varying morphological constitutions. One hundred and thirty students were measured by Behnke's method in order to classify them as obese, muscular, or lean. Within each classification there were two groups, active and inactive. The training period was 10 weeks long with the active subjects exercising on the treadmill 3 times per week. Cholesterol was measured before and after the training period. The initial mean of all the active groups was 189 mg./100 ml. of blood. The same value for all of the inactive groups was 196 mg./100 ml. of blood. Each training session consisted of six 5-minute bouts of exercise on the treadmill with a 5-minute rest between each. Upon completion of the training period the active group's mean level was found to be 184 mg./100 ml. of blood, whereas the same level for the inactive groups was 200 mg./100 ml. of blood. Campbell reports that the obese active group was the only group to show a significant change in serum cholesterol.

Campbell (1967) used a large group of healthy male college students to study cholesterol concentrations during physical training and subsequent detraining. The subjects were initially classified according to morphological configuration and separated into three groups: obese, muscular, and slim. The two treatments were 10 weeks of conditioning on a treadmill three times per week and 10 weeks of

deconditioning with no activity. Blood samples for measuring cholesterol concentrations were taken before training, after Treatment I, and after Treatment II. Obese subjects had the highest initial levels and showed the greatest decrease during activity and greatest increase once activity ceased. The results suggested that when attempts are made to appraise the influence of physical activity on serum cholesterol concentrations, the body configurations of the subjects must be considered.

Holloszy (1964) studied the effects of a 6 month program of endurance exercise on the serum lipids of 15 professional men whose ages ranged from 35 to 55 years. All of these men had led basically sedentary lives for 3 or more years. The exercise program consisted of progressively more strenuous endurance calisthenics and distance running on an average of 3-1/2 times per week for 6 months. At regular intervals the physical condition of the subjects was determined. Blood samples were taken three times before the program was begun for serum cholesterol determinations, once a month during the study, and again three times during the last week of training. During the training period the subjects were asked not to alter their work habits or diet. A detailed dietary history was obtained from each subject at the beginning and end of the study and each subject was shown how to keep detailed records of food intake. Body weights were also determined before, during, and after the study.

The results of the study indicated that there was a significant increase in the length of time the subjects were able to run on a treadmill at a rate of 7 miles per hour up to 8.6% incline. The

average caloric intake reported by the subjects did not change significantly and the average body weight also remained constant, in spite of an estimated average increase in caloric expenditure resulting from the exercise program of approximately 1,000 calories per week. This might suggest that there was a transfer of body mass from adipose tissue to muscle. No significant changes were revealed for the cholesterol values of the group. Reductions in serum cholesterol levels were noted only in those subjects who were losing weight, and the serum cholesterol returned to higher levels when the body weight stabilized. Two subjects who gained weight had significant increases in serum cholesterol.

Gsell and Mayer (1962) conducted a study on the population of a Swiss Alpine village named Blatten. The authors compared serum cholesterol concentrations in about 300 city dewllers of Basel with those of 300 Blatten residents. Blatten was described as situated at 5,057 feet in a remote valley of the southern part of the Alps. The inhabitants supported themselves by farming performed under extremely difficult conditions. Until 1955, the village was separated by several miles from any roads suitable to automobiles, trucks, or carts. Farming areas were spread on steep slopes varying in altitude from 4,000 to 8,000 feet. All distances had to be walked as mules were expensive and rare. Men frequently carried loads of 100 pounds or more and loads up to 60 pounds had been seen to be carried by elderly women.

By comparison, the serum cholesterol levels of both men and women at various ages were consistently lower in Blatten than in

Basel. Also, there was greater longevity in the Blatten group. It was noteworthy that 27% of the total caloric intake of Blatten residents was derived from animal fats, involving dairy products, largely composed of saturated fatty acids. The saturated fatty acid intake of the Basel population was definitely less. The differences in serum cholesterol were not accounted for by differences in weight, adiposity, altitude, climate, or smoking habits. The authors suggested that physical activity was the primary factor accounting for the difference in serum cholesterol concentrations. Although the effect of exercise on blood cholesterol seems to vary with different population groups, it does appear that serum cholesterol levels can be lowered by exercise, irrespective of certain dietary factors.

Stulb and others (1965) carried out a study on 26 pairs of white men living in Evans County, Georgia. The subjects were matched on the basis of age and classified according to a higher or low serum cholesterol value. The 52 paired subjects were drawn from a much larger population on the basis of their fulfilling the above requirements. The subjects were then divided into two groups; Group I which was the low serum cholesterol group and Group II which was the high serum cholesterol group. The initial mean serum cholesterol Level for Group I was 141.9 mg./100 ml. of blood, whereas the same mean for Group II was 281.3 mg./100 ml. of blood. These measurements were taken in the fall of the year. At this time also, each subject was interviewed in his home to establish the nature and amount (calories) of his normal daily food intake. Exercise was graded according to the type of occupation. Laborers and farm sharecroppers

were classified as high exercise; proprietors, professionals and large farm owners were classified as low exercise; and all others, such as service workers and tradesmen, were classified as middle exercise.

The serum cholesterol tests and food intake interviews were repeated again in the spring of the following year. Group I's mean serum cholesterol level had increased to 158.9 mg./100 ml. of blood, whereas Group II showed a decrease to 274.1 mg./100 ml. of blood. The results disclosed no significant relationships between serum cholesterol and dietary components. On the other hand, a highly significant inverse relationship between exercise and cholesterol emerged. Significant seasonal differences in dietary constituents were observed which consisted essentially of a decreased caloric intake during warmer months. The authors did note the possibility of a relationship between the seasonal dietary pattern and the seasonal cholesterol pattern but the data were inadequate to test the relationship possibilities.

Several valid studies have been focused on the lipoprotein profiles exhibited by elite runners who have exercised at high levels for at least several years. The resultants consistently revealed low levels of plasma triglycerides, LDL, VLDL, cholesterol and elevated HDL.

The Stanford Heart Disease Prevention (1977) analysis of lipoprotein in runners as compared to a control group also revealed lower cholesterol, triglycerides, LDL, VLDL, and higher HDL. A higher dietary intake of polysaturated fats and smoking did not bear a relationship on the lipoprotein levels.

The Lipid Research Clinic of Iowa City conducted a recent study of 2,874 school children. Coronary mortality determined from death certificates was increased in the young relatives of school children with the High Group Choesterol indexes. Stroke mortality was higher, although not significantly in the older relatives of the high index cases. The study concluded that hypercholesteremia in children identified families at risk for CHD (Schrot, 1975).

HDL Lipoproteins and CHD

Persistent interest in HDL has greatly intensified in recent years, stimulated by the finding that HDL is inversely related to coronary artery disease. Clinical epidemiologic observations of a striking, persistent and independent negative association between HDL levels and coronary vascular disease have, in turn, generated new interest in structure, composition and metabolism of this fascinating lipoprotein. HDL is available in plentiful amounts from normal human plasma and is readily separable in its lipid and apoprotein components. The protein moiety of HDL is heterogeneous. The two A apoproteins, apo A-I and apo A-II, constitute about 90% of HDL protein. The ratio of apo A-I to apo A-II is about 3:1. Apo A-I is almost completely absent in patients with Tangier disease (Familial High Density Lipoprotein Deficiency), while the amount of apo A-II is reduced to 6% of normal. Apoprotein C, the main apoprotein of VLDL, constitutes about 5% of HDL protein mass. At least three different peptides belong to the C family of apoproteins. C-I, C-II, and C-III. Apoprotein D, also known as the "thin-line peptide," and apoprotein E or the arginine-rich apoprotein, are other minor constituents of

HDL. The unique features of the HDL protein components and their role in lipid metabolism are the subject of prolific investigation.

The major HDL apoproteins, apo A-I and apo A-II, can be readily isolated from human HDL and are easily separated by various chromatographic techniques. There exists an uncertainty today about the relative contribution of the liver and intestine to HDL synthesis. It remains to be demonstrated whether the majority of HDL arises as a "nacent" lipid-poor form from the liver or a delipidated chylomicron remnant from the intestine. Similarity, the relative contribution of these organs to plasma A-I and A-II apoprotein levels is unclear. Also unclear, is the site of HDL degeneration, though liver and kidney lysosomes have been incriminated (Levy, 1978).

Little is known about HDL function(s). HDL may be an important factor in cholesterol efflux from the tissues, thereby reducing the amount of cholesterol deposited there. Alternatively suggested is the theory that HDL may pick up cholesterol esters and phospholipids during normal VLDL lipolysis in the plasma, a property which theoretically may be linked to current postulates that HDL may play a protective role in atherosclerosis.

HDL particles act as donors of apo-C to chylomicrons and VLDL; a specific apo-C, C-II, activates the enzyme lipoprotein lipase for their degradation and removal (AHA Abstract, 1979). HDL may also play a role in triglyceride metabolism. The extent to which HDL is involved in triglyceride metabolism beyond its role as a carrier of apo C-II is unknown. HDL levels are markedly decreased in subjects with exogenous hypertriglyceridemia, and HDL apoprotein metabolism is

enhanced by the increased triglyceride flux in patients with nephrotic syndrome as well as in normals in high carbohydrate diets (80% of calorie intake). Why this occurs is unknown. HDL levels are clearly lower in humans than in animal species relatively resistant to atherosclerosis, i.e. the dog, sheep and rat (Havel, 1979).

HDL levels are relatively insensitive to diet, increasing somewhat with weight reduction, hypertriglyceridemic subjects or with moderate increases in alcohol intake. HDL levels are not affected by any of the current hypolipidemic drugs. Cholestryamine and D-thyroxine all manifest their effects on VLDL and LDL, as does the surgical technic of ileal bypass, but none of these effect HDL concentration (Levy, 1978).

Attention has focused on the lipoprotein profiles exhibited by 20 elite runners, eight good runners, and selected age-matched nonrunning controls at the Stanford Heart Disease Prevention Center (Martin, Haskell and Wood, 1977). A significantly higher HDL cholesterol was found in all groups of runners versus their control. It was interesting that the values for HDL cholesterol increased with increasing age in active runners while the controls showed a slight decrease or no change in HDL. However another group found no difference in serum HDL concentration between sedentary and "high physical activity" subjects (Mjos, 1977). Hartung et al. (1980) investigated the effect of diet on HDL cholesterol in 59 healthy middle-aged marathon runners, 84 joggers and 74 inactive men. Meat consumption was not significantly correlated with HDL. Results suggested that HDL differences (marathon runners, 65 mg. per

deciliter, joggers, 58 mg. per deciliter, inactive men 43 mg. per deciliter) among the three groups were primarily the result of distance run, not dietary factors. Distance run was also the best predictor of the HDL total cholesterol ratio and of total cholesterol (a negative correlation), and it was second only to weight in predicting triglyceride levels.

Investigators in five centers in the United States analyzed data collected on HDL-cholesterol in men and women aged 40 years and older, as part of a long-term study of plasma lipoproteins and coronary disease in free-living populations. They reported that HDL-cholesterol levels were consistently lower in individuals who had clinical CAD than in those who did not, and, based on multivariate analysis, the lower HDL-cholesterol levels could not be explained by associations with other factors, such as plasma triglyceride level (Castelli, 1977).

Investigators of the Framingham Heart Disease Epidemiology Study reported that the risk of developing clinical CAD in a four-year period in men and women aged 49-82 years was more strongly related to initial HDL-cholesterol levels than to levels of LDL-cholesterol (Gordon, 1977). Similar studies were reported for men aged 20-49 years from the Tromso Heart Study in Norway (Miller, 1977). Jenkins, Harper and Neshel (1978) reported, in a series of patients studied by coronary angiography, an inverse relationship between severity of luminal narrowing and HDL-cholesterol levels.

Exercise and CAD

Morris et al. (1953) undertook an epidemiological study of

coronary disease with relationship to physical activity and inactivity of work. Transport workers, postal workers and clerks were observed. Bus conductors were found to have less coronary heart disease than bus drivers, and postmen less than telephonists, executive officers, and clerks. Moreover, what disease the conductors and postmen had was less. More active men, as classified by occupational and leisure exercise habits, seem to be less subject to coronary heart disease (Currens, 1961; Shane, 1966; Fox, 1969).

Pollock randomly placed 19 men into two groups for the purpose of investigating the effect of frequency of training on working capacity, body composition, and circulo-respiratory measures. The subjects trained 30 minutes per day for 20 weeks. Group I trained two times per week whereas Group II trained four times per week. The training program consisted of continuous walking, jogging, or running. Pretests were administered for the various parameters measured as well as posttests. The results showed both groups improved significantly in maximum VO_2 and significantly decreased the values for resting, exercise, and recovery heart rates. The body composition for Group I stayed the same but Group II significantly improved in total body weight and percent fat. The analysis between the groups showed that improvements were manifested in accordance with frequency of participation (Pollock, 1967).

Paffenbarger at the University of California School of Public Health, studied San Francisco longshoremen with sophisticated techniques and compiled a mortality study of coronary heart disease in various grades of these dockers on the basis of the physical exertion

involved in their jobs (Paffenbarger and Hale, 1971). A total of 6,351 longshoremen 35 to 74 years of age were enrolled and followed for job changes and coronary mortality during the years of 1951 to 1972. During this 22 year follow-up observation, 598 longshoremen died from coronary heart disease. Sixty-six deaths were charged to the heavy work category, 107 to the moderate and 425 to the light category. Also, the data on sudden death support most plainly the protective threshold implications of energy output. Taking the sudden-death rate from coronary disease for heavy-working men as 1.0, the relative risk among those in moderate work is 3.5, and those in light work is 2.8. Therefore, vigorous physical exercise, as defined by an apparent threshold or critical level of energy output, is associated with reduced risk of coronary mortality, particularly the sudden death syndrome. These findings consistently support a hypothesis that protective rather than merely selective influences account for the observed reduction or deferment risk of coronary heart disease.

Paffenbarger also followed these longshoremen to note any changes in their occupations (to answer the question as to whether less fit men take a series of jobs requiring less and less physical exertion). They were placed into three grades. The heavy workers had coronary mortality rates substantially below those of the other groups. Paffenbarger approached the problem of selection between the groups. Strikingly enough, there was no significant differences between the groups with respect to smoking, blood pressures and cholesterol levels; thus, he concluded that the differences in

coronary mortality could not be accounted for by selection with respect to these factors.

Another interesting study has been conducted by Paffenborger (n.d.). He made contact with all the 16,000 Harvard graduates over the course of many years, and obtained a medical history, and a history of their student existence including their sport activities. He also asked about any continuing, athletic activity. He demonstrated that if an athlete at the University continued his sport, this seemed to confer a protective effect against the development of coronary diseases; however, he was not protected against heart disease in middle age any more than men of the same age who took the same amount of exercise and who were not athletes as students. If a student did not continue to take regular exercise, his student activity seemed to exert no subsequent protective effect with respect to coronary heart disease.

Morris (1979) studied 17,000 middle-aged civil servants, of the executive grade, an intermediate grade between clerical and administration. They were all sedentary office workers. These men filled in a detailed five minute questionnaire on how they had spent an unannounced Friday and Saturday. Where men were matched having their first coronary against matched controls, similar age, etc., the men suffering their first coronary were much less likely to have taken any vigorous exercise on the two days studied than their matched controls.

The only type of activity appearing to have much importance was physical activity involving a caloric expenditure of greater than

7.5 kilocalories (equivalent to heavy industrial work). The difference in mortality one year later from coronary disease was 1.0 for the exercise groups and 4.3 for the others. On 500 of these men, the ones reporting vigorous physical exercise showed fewer ECG changes suggestive of ischemia, than their less vigorous controls: 4.8% against 10.4%. An interesting striking difference existed in the frequency of ectopic beats between these two groups: 2.2% against 7.1%.

Fox (1969) presented, in the American Journal of Cardiology, a thorough review of much of the recent work done by other authors on the subject of physical activity in relation to coronary heart disease. Dr. Fox concludes that the following statements seem to be warranted in regard to recent work in this area:

1. The more active men in most study cases are less subject to coronary heart disease, even less to myocardial infarction, and much less to sudden death. The data refer to both occupational and nonoccupational physical activity.

2. The major influence of physical activity appears to relate to myocardial infarcts, scars and fibrous patches, less to coronary occlusion and least to coronary atherosclerosis.

3. The intensity and duration of physical activity does not differ greatly among groups with significantly different manifestations or coronary heart disease. It appears that the amount of physical activity engaged in by the more active groups would be acceptable to many presently sedentary citizens. One might be able to increase the intensity and shorten the duration and obtain the same

suggested benefit in an individually acceptable prevention regimen.

4. More work is needed to prove the effectiveness of the suggested preventive approach through increased habitual physical activity, and to refine prescriptions of activity for patients and to enhance motivation.

5. Apart from the important need to acquire more data to prove the effectiveness of habitual physical activity, Dr. Fox mentions the growing acceptance of physical conditioning and reconditioning as a means to help overcome the depression and anxiety that results from coronary heart disease. He states that it is prudent to include habitual physical activity in a program to prevent or manage nonacute coronary heart disease.

Diet: Effects on Lipids and CHD

In 1960, an executive committee on Diet and Heart Disease was established to conduct a pilot study to test the hypothesis that alteration of amount and type of fat and amount of cholesterol in the diet would decrease incidence of first attacks of clinical coronary heart disease in middle-aged men. The committee concluded that such a study was both feasible and necessary.

The study population was drawn from five "open" centers across the United States (Baltimore, Boston, Chicago, Minneapolis-St. Paul, and Oakland) and one "closed" center (Faribault State School and Hospital, Minnesota). Approximately 100,000 men were included in the 6-year study. The study tested principally three double-blind diets: (1) The control diet contained 40% of calories from total fat, 18% or more from saturated fat, 5% or less from polyunsaturated fat and an

average daily cholesterol intake of 650 to 750 mg. (2) One experimental diet provided reduction in total fat to 30% of total calories, saturated fat to less than 9%, cholesterol to 350 to 450 mg. per day, and an increase in polyunsaturated fat to 15%. (3) A second experimental diet provided total fat at 40% of total calories, reduction of saturated fat to less than 9%, cholesterol to 350 to 450 mg. per day, and an increase in polyunsaturated fat to 20%.

In the open-study centers, the fall in serum cholesterol was 25 mg./100 ml. of blood and 28 mg./100 ml. of blood on the two experimental diets, while in the closed institution the mean falls were 36.2 mg./ 100 ml. of blood and 31.3 mg./100 ml. of blood. The difference reflected a better adherence to the experimental dietary programs in the closed population. It was of interest to note that the largest drop in serum cholesterol was experienced by men with the largest decreases in weight and the highest initial cholesterol levels. The study clearly points out that regulation of the diet can influence the level of serum cholesterol. Caloric control, then--reducing the obese and keeping them reduced--is of great importance, along with modification of the fat-cholesterol composition of the diet (The National Diet-Heart Study, 1968).

West and Hayes (1968) compared the serum cholesterol levels of 233 nonvegetarians with those of 233 vegetarians who had been matched for age, place of residence, sex, marital status, height, weight, and occupation. The vegetarians were drawn from a larger population of Seventh-day Adventists who, as part of their religion, believe in refraining from eating meat, fish, or fowl. The results indicated

that the differences between the serum cholesterol levels of the two groups was statistically significant at the .01 level of confidence. Several degrees of nonvegetarianism were noted, and evidence was clear that as the degree of nonvegetarianism increased, the levels of serum cholesterol also increased.

Goode (1966) studied the effects of exercise and a cholesterol-free diet on human serum lipids. Six male subjects whose ages ranged from 25 to 46 were maintained on a diet free of all animal fat for a period of 54 days. For the first 3 weeks the subjects underwent normal activities and basal measurements for cholesterol were attained. After 3 weeks, three of the subjects began running on a treadmill for 25 minutes per day for 14 consecutive days while the other three subjects acted as controls. There was a 5-day rest period following this and then the procedure was reversed for another 14-day period. In the first 3 weeks of the restricted diet all subjects showed substantial reductions in serum cholesterol. Then as the experiment continued, the cholesterol levels tended to increase in both groups. Exercise can initiate a transient increase of serum cholesterol in subjects on an unrestricted diet. This is thought to be due to increased fat mobilization for energy production, which could in turn be a first step toward lowering of body stores of cholesterol.

The Finnish Mental Hospital Study was undertaken as a dietary intervention trial to test the hypothesis that there is a close correlation between the consumption of saturated fats and the mortality from coronary heart disease. Practically total replacement

of dairy fats by vegetable oils in the diets of these hospitals was followed by a substantial reduction in the mortality of men from CAD. Total mortality also appeared to be reduced. As to the causes of death other than CAD, none was significantly influenced by dietary change (Turpeinen, 1979).

The Inter-Society Commission for Heart Disease Resources (1970) speaks of a "habitual diet high in saturated fat-cholesterol-calories as a risk factor in CAD. The National Diet-Heart Study subscribes to the view that diet is a "key factor in the etiology of atherosclerosis" and suggests that the disease can be prevented by diet."

Studies have shown associations between fat intakes and blood-cholesterol levels on a national or community basis. Within groups in developed communities, however, no correlations between fat consumption and cholesterol levels have been reported on an individual basis.

Morris et al., 1963, found no relationship between individual levels of fat (or other nutrient) intake and individual blood-cholesterol levels.

Few prospective studies have reported results directly relating individual dietary intake to the later development of CAD. However, the Framingham Study, 1970, collected detailed dietary data which were correlated with subsequent CAD incidence. Neither study found associations between dietary fat intake and CAD incidence.

Emotional Stress and CAD

Over the past few years there has been more acceptance of the

importance of emotional tension as a likely contributor to not only acute coronary events, perhaps through dysrhythmias in a heart made vulnerable through sympathetic stimulation and an increase in catecholamines, but also as a part of other basic mechanisms accelerating angina.

Osler (1896) was perhaps the first to suspect that emotional factors were directly related to coronary heart disease. Making observations from case studies, he stated that: ". . . the worry and strain of modern life . . . and . . . the high pressure at which men live, and the habit of working the machine to its maximum capacity" (p. 800), "are the causes of arteriosclerosis and especially of coronary disease." He observed that arterial degeneration was not only very common but also was developing at a relatively "early age." In 1910, Osler reported that the most important results in treating coronary heart disease may follow a change in a man's habits of life. He prescribed that his patients go slowly and further stated, that: "a man who has kept a full head of steam in his boilers must learn to stoke the engines in due proportion to the work expected" (p. 976).

According to Richardson (1896), sufferers of angina:

> . . . were middle aged men who had led a busy, often comfortable, and vigorous outdoor life. They were active men and as a rule laborious and troubled men . . . who had felt they had had a great deal to account for. They had been over ambitious and had failed to get all they wished . . . and had been perpetually harassed with difficulties they did not feel able to overcome, or did not feel capable of resigning to fate. (p. 292)

Richardson suggested that his patients "be freed from excitement, from passion, from fear and from everything that causes fatigue and

what is called nervous exhaustion" (p. 294).

According to Russek (1959), prolonged emotional strain associated with job responsibility preceded the attack of coronary disease in 91% of his series of 100 patients. He later reported (1967) that the most characteristic trait of the young coronary patient was his restlessness during leisure hours and his sense of guilt during periods of relaxation. As a consequence, the coronary heart disease patient rarely took vacations and frequently participated in a regimental series of obligatory activites. Furthermore, Russek recognized that emotional stress promoted fatigue which frequently contributed to the failure to achieve daily exercise, creating a sense of time urgency and decreased motivation. Emotional tension was also recognized as a precursor to compulsive eating, drinking and smoking in many people as compensation for anxiety. These habits were directly related with several of the recognized risk factors in coronary heart disease of hypercholesterolemia, overweight, hypertension, lack of exercise, and cigarette smoking. Russek concluded that the indirect effects of emotional stress, barring all other effects, must elevate it to a position of considerable significance in the etiologic picture of coronary heart disease.

Friedman and Rosenman (1971) are the researchers who conceptualized the Type A-Type B Behavior Patterns. Through a study of the pertinent literature beginning in 1955, they located enough published evidence that suggested the possible presence of various emotional and stress patterns identified with the coronary heart disease patient. To summarize, the pattern is described by these

investigators as

> a characteristic action-emotion complex which is exhibited by those individuals who are engaged in a relatively chronic struggle to obtain an unlimited number of poorly defined things from their environment in the shortest period of time and, if necessary, against the opposing effects of other things or persons in this same environment. (Friedman, 1969)

Individuals who manifest this behavior pattern to a greater degree are called Type As, whereas those who tend to show the opposite pattern of relaxation, serenity, and lack of time urgency are designated Type Bs.

Research indicates an association between occupational rank and Pattern A (Rosenman et al., 1964; Rosenman, Friedman, Straus, Jenkins, Zyzanski & Wurm, 1970). Individuals in professional and managerial occupations tend to have a higher frequency of Pattern A behavior than individuals at lower levels of the occupational hierarchy.

One of their early studies (1958) examined the possible effects of emotional stress upon the serum cholesterol and blood clotting time of 42 volunteer male accountants. Subjects were selected due to the unique phasic variations in their work loads and its association with deadline work periods. In 83% of the entire group, the maximum cholesterol was observed during times of their maximal stress (workload and deadline). In 76% of the entire group, the individual's minimum observed cholesterol value occurred at the times of their least stress. Similar results were obtained on blood clotting time which shortened from an average of 9.4 minutes during minimum stress periods to 5 minutes at the time of maximal work stress. It was concluded that temporal periods of unusual emotional stress and tension are associated with a sudden and often

profound increases of serum cholesterol and acceleration of blood clotting time.

In 1959 Friedman and Rosenman began their research on the differences in behavior patterns. Three groups were selected according to the behavior patterns which they habitually manifested in their work. Eighty-three "As" and 83 "Bs" were grouped along with a group "C" consisting of 46 unemployed blind men selected as manifesting a chronic state of insecurity and anxiety. These groups were compared with respect to their serum cholesterol levels, blood clotting times, presence of clinical coronary heart disease and presence of arcus senilis. With all controllable variables comparable it was observed that the average serum cholesterol level was higher in the "A" group while clinical coronary heart disease was seven times more frequent and arcus senilis three times greater than in the "B" or "C" groups. These results suggested that the behavior pattern of the "A" group was of itself largely responsible not only for higher serum cholesterol but also for markedly increased incidence of both clinical coronary artery disease and arcus senilis.

The Western Collaborative Group Study (WCGS) was conducted as a double-blind prospective investigation in which the researchers rating the behavior pattern had no knowledge of other risk factors and did not participate in subsequent diagnosis of the presence or absence of CAD. The responsibility for diagnostic judgments was vested in two cardiologists both of whom worked independently of the study and had no knowledge of the behavior-pattern classification and of the presence or absence of other risk factors. Of the 3,524 men,

aged 39 to 59 years at intake, 3,154 completed participation in the longitudinal study. All were employed in 10 California companies. The following kinds of data were obtained: medical and socioeconomic histories; dietary and smoking habits, blood pressure; serum cholesterol, triglycerides, and lipoproteins; blood clotting times; and anthropometric measurements. These data were obtained at intake and annually until the study was terminated, providing 8 to 9 years of follow-up.

Men judged at intake to be Pattern A had more than twice the rate of new CHD during 8-1/2 years as men originally judged to possess Pattern B behavior. Of approximately 1,500 men classified as Pattern A, 178 developed clinical CHD 8-1/2 years later. Only 79 of the some 1,500 men diagnosed as Pattern B developed CHD during the period of the prospective study. The results also showed that Pattern A subjects with CAD were 5 times more likely to have a second myocardial infarct than were Pattern B subjects with CAD.

Friedman and Rosenman (1969) and Rosenman and Friedman (1961) showed that the average serum cholesterol levels of men with fully developed Pattern A behavior were significantly higher than those of their fully developed Pattern B counterparts. While Friedman et al. (1968) were unable to replicate these findings, Blumenthal et al. (1966) reported a similar difference for the 39-49 year old age group at intake in the WCGS, and Jenkins, Zyzanski, and Rosenman (1973) present indirect evidence linking Pattern A, as measured by the Jenkins Activity Survey (JAS) with increased levels of serum cholesterol.

Rahe and his colleagues (Rahe, Clark & Arthur, 1970), have conducted an extensive series of studies of the association of human subjects' psychological states with their serum concentrations of cholesterol. Most of the research used Navy personnel exposed to a variety of stressful training situations; the group included underwater demolition trainees, submariners, or naval aviators. Elevated serum cholesterol levels were observed when subjects felt overburdened by demands of the training (for example, during exams and periods of learning new skills); when subjects reported feeling depressed, angry, fearful, and lethargic; and, when there was a threat of eminent failure.

Further support for the association between serum cholesterol level and psychological stress comes from a study by Friedman, Rosenman, and Carroll (1958). These investigators studied a group of accountants who agreed to be bled twice monthly for approximately 6 months beginning in the first month of the year. For at least 1 week prior to April 15, the final date for tax returns in the U.S., the accountants were subjected to the stressful experience of finishing all the tax forms they had contracted to complete. Many of the accountants were also subjected to the same type of stressful experience in January, when tax inventories had to be prepared for various corporate clients. Along with serum cholesterol determinations taken twice monthly during the 6-month period (January through June) the accountants each kept a dietary history for the week of April 2 to 9 and again on May 14 to 21.

The results showed that during the first two weeks of April,

serum cholesterol levels were significantly higher than during February and March. The average cholesterol level in April for the accountants who were also engaged in estimating January tax inventories was higher than the level for those who did not have to work on the inventories. No change was noted in the diets of any of the accountants during the periods in which cholesterol rose. The average cholesterol level of all accountants fell sharply after April 15. There were also several experiments antedating the 1958 publication showing that cholesterol levels are appreciably higher during periods of stress than at other times (Mann & White, 1953). Most of this research used university or medical school examinations as the stressor stimulus and the period of regular academic work as baseline.

Another dimension was found in the studies of Brooks and Mueller (1966), Rahe et al. (1968), and Wolf et al. (1962). They found that "active" psychological states such as motivation to master events accompany low levels of serum cholesterol and high levels of serum uric acid. Tasks and responsibilities assumed as an expression of one's own drive in men with high urate levels were not experienced as unduly burdensome or unpleasant, in contrast to the sensation of being driven associated with high cholesterol levels in medical students at examination time or accountants at tax deadline time. In contrast, Nixon (1976) found that hyperuricaemia occurred along with hypercholesteralemia in aggressive individuals who had driven themselves into ill-health with morbid arousal.

There appeared to be no published studies directly associating

hypertension with Pattern A. Indeed, there is evidence from at least one study (Skekelle et al., 1976) which indicated that prevalence of hypertension is unrelated to Pattern A in men. On the other hand, the WCGS suggested that Type A men at intake had higher diastolic blood pressure than Type B men (Rosenman et al., 1966). Of greater significance, perhaps, is the finding in the 2-1/2-year follow-up that elevated diastolic blood pressure (exceeding 95 mmHg) significantly enhanced the risk of CAD only when this factor occurred in Pattern A subjects (Rosenman et al., 1966). There are a host of experiments showing that psychological stimuli indicative of Pattern A produced episodic rises in blood pressure (Hokanson et al., 1963; McGinn et al., 1964), but it is not at all certain that such elevations are closely related to coronary disease (Graham, 1972; Gutmann & Benson, 1971).

Prediction of Energy Expenditure from Walking

Reference has been made in the literature to the fact that energy expenditure and gross body weight are significantly related during walking and running (Bobbert, 1960). Van der Walt and Wyndham (1973) developed an equation for calculating VO_2 from walking by calculating linear regression of energy expenditure during walking as a function of mass (M) and speed (V). Oxygen consumptions and stride frequencies were measured on six subjects at four different speeds of walking and running. Measurements of leg length were also made and pace lengths were calculated from stride frequencies. Correlations of 0.85-0.99 between mass and oxygen consumption were highly significant at all levels of walking and running. This study demonstrated

however, that pace length is not an important factor in the prediction of VO_2 during running and walking if it is chosen naturally by the subject. There is no point therefore in introducing pace length into the equation for predicting VO_2. The energy expenditure of both walking and running can be predicted by an equation which has the same form: $VO_2 = A_1 + A_2M + A_3\ MV$ where A_1, A_2 and A_3 are regressive constants.

Summary

Investigations generally suggest that a population with a high average level of serum cholesterol tend to have a high reported mortality from CAD. Conversely, lower death rates and lower atherosclerosis at postmortem have been reported from areas where substantially lower indigenous cholesterol levels have been found. Marathon runners have demonstrated lower levels of cholesterol triglycerides, LDL, VLDL and higher HDL and serum triglycerides have been lowered in hyperlipidemic individuals with a one-time exercise session. Interest in HDL lipoprotein has greatly intensified in recent years, stimulated largely by the finding that HDL is inversely related to CAD. Friedman and Rosenman's work on the Type A personality has provided impetus for the model of behavior which is characterized by time-urgency hard-drivingness. These individuals displayed higher lipid levels and a greater incidence of CAD. Dietary contributions to hyperlipidemia has been of some dispute but the most current concensus does not define a statistically significant correlation between polysaturated fat intake and hyperlipidemia. Jobs which involved active physical

activity vs. sedentary jobs usually are associated with a lower incidence of CAD.

CHAPTER III

THEORETICAL FRAMEWORK

A generally accepted theory is that the defense reaction, or the "flight or fight" response is a basic physiologic stress response which is activated by a wide range of stimuli and involves a wide range of cardiovascular changes for which the integrating area appears to be, at least in part, the hypothalmus. This concept has been established by a number of investigators including Hilton (1966) and Folkow (1966). Research has demonstrated that the cardiovascular responses elicited by stimulating this portion of the brain stem mimic cardiovascular responses observed in man during mental stress.

Since periodic chronic stimulation of the defense area will lead to hypertension, it is reasonable to assume that periodic emotional or mental stress in man may lead to the same result. All the cardiovascular effects observed due to activation of the automomic nervous system during stress are accompanied by a constellation of hormonal responses designed to provide feedback circuits and to direct the overall system toward a primary goal of maintenance of an intact system (Abbey, 1972). During the initial response to a stress, adrenal medullary catecholamines tend to potentiate the sympathetic responses as well as to mobilize glucose and fats as energy substrate. Aldosterone from the adrenal cortex and

antidiuretic hormone released from the posterior pituitary tend to conserve sodium and water. The anterior pituitary is also activated and releases a number of hormones including growth hormone, prolactin and adrenocorticotrophin (ACTH). The first two hormones probably provide further substrate for tissue metabolism by causing the breakdown of adipose stores and by suppressing the capacity of skeletal muscle to use glucose as a substrate. The latter function acts to conserve glucose for use by the brain and by blood cells. ACTH activates the secretion of glucocorticoids from the adrenal cortex. The exact functions of the glucocorticoids in providing resistance to stress are uncertain although effects on energy substrate availability, tissue metabolism and tissue reactions to injury and inflammation may be important aspects of their functions (Auefton, 1796, pp. 1026-1028).

Increases in blood lipids due to these changes in the internal chemical environment of the body may develop in response to a state of exhaustion caused by prolonged and excessive arousal. It is the natural mechanism for meeting physical challenges--either fighting or running away--for involvement in the pecking order of an organization, and for the display of appeasement maneuvers.

Cholesterol, is present in the diet of all persons, and it can be absorbed slowly from the gastrointestinal tract into the intestinal lymph. It is highly fat soluble, but only slightly soluble in water and it is capable of forming esters with fatty acids. Approximately 70% of the cholesterol of the plasma is in the form of cholesterol esters.

Besides the cholesterol absorbed each day from the gastrointestinal tract, which is called "exogenous cholesterol," a large quantity, termed "endogenous cholesterol" is formed in the cells of the body. Essentially all of the endogenous cholesterol that circulates in the lipoproteins of the plasma is formed by the liver, but all the other cells of the body form at least some cholesterol which is consistent with the fact that many of the membranous structures of the cells are particularly composed of this substance. As illustrated by the formula of cholesterol (Figure 1), its basic structure is a sterol nucleus. This is synthesized entirely from acetyl Co-A. In turn, the sterol nucleus can be modified by means of various side chains to form (a) cholesterol (b) cholic acid, which is the basis of the bile acids formed in the liver, and (c) several important steroid hormones secreted by the adrenal cortex, the ovaries and the testes.

By far the most abundant use of cholesterol in the body is to form cholic acid in the liver. As much as 80% of the cholesterol is converted into cholic acid. This is, in turn, conjugated with other substances to form bile salts, which promote digestion and absorption of fats. A small quantity of cholesterol is used (a) by the adrenal glands to form adrenocortical hormones, (b) by the ovaries to form progesterone and estrogen, and (c) by the testes for form testosterone. A large amount of cholesterol is precipitated in the corneum of the skin. This, along with other lipids, makes the skin highly resistant to the absorption of water-soluble substances and also to the action of many chemical agents, for cholesterol and the other

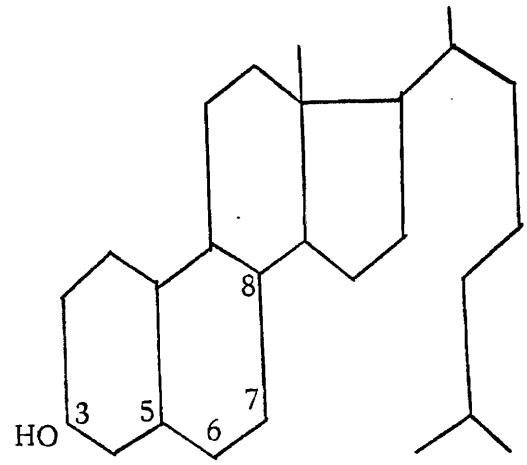

Figure 1. Structural formula for cholesterol.

lipids are highly inert to such substances as acids and different solvents that might otherwise easily penetrate the body (Guyton, 1976, pp. 924-925).

The triglycerides are used in the body mainly to provide energy for the different metabolic processes; this function they share almost equally with the carbohydrates. The basic structure of the triglyceride molecule is illustrated in Figure 2. An average of 30 to 50% of the carbohydrates ingested with each meal is converted into triglycerides, then stored and later used as triglycerides for energy.

Whenever a greater quantity of carbohydrates enters the body than can be used immediately for energy or stored in the form of glycogen, the excess is rapidly converted into triglycerides and then stored in this form in the adipose tissue. Most triglyceride synthesis occurs in the liver, but smaller quantities are also synthesized in the adipose tissue. The triglycerides that are formed in the liver are then mainly transported by the lipoproteins to the adipose tissue where they too are stored until needed for energy (Guyton, 1976, pp. 916-921).

In the postabsorptive state, over 95% of all the lipids in the plasma (in terms of mass, but not in terms of rate of transport) are in the form of lipoproteins. These are transmitted as spherical macromolecular complexes, wherein an inner core of hydrophobic lipids (triclyceride and cholesterol esters) is encased by a membrane of unimolecular thickness consisting of various proteins (apoliproteins, or simply aproproteins) in association with hydrophillic lipids (free

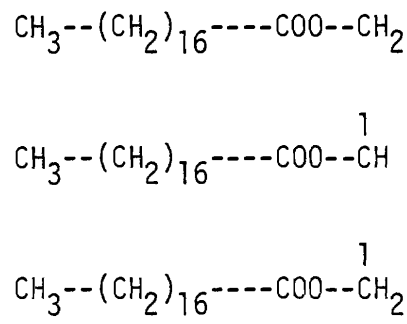

Figure 2. Basic structure of the triglyceride molecule.

cholesterol and phospholipids).

Specific differences among the various lipoproteins classes involve how much they contain of each of four types of lipids, which affects their size and density, and the nature of the apoprotein in their membrane. These differences allow classification of lipoproteins on the basis of ultracentrifugal density, with those containing mostly triglyceride being termed "very low density lipoproteins (VLDL)" and those with predominately cholesterol called "low density lipoproteins (LDL)"; when the total lipid content is slightly less than the weight of protein in the membrane, the density is "high (HDL)."

When classified on the basis of their mobility on paper electrophoresis, LDL are termed betaliproproteins, VLDL are prebetaliproproteins, and HDL are called alphaliproproteins. These three classes of liproproteins are normally present in fasting sera. A fourth class, normally present only after ingestion of fat, are called chylomicrons. These are of such low density that they float even without centrifugation, and, because of their large size and proportionately low protein content, they fail to migrate on paper electrophoresis, remaining at the origin.

Research suggests that the arterial wall intima is permeable to small molecular complexes in inverse proportion to their size. In addition, elastin, a component of arterial wall, has a demonstrable affinity for aproprotein B, which is present on all liproproteins except HDL. Accordingly, small lipoproteins such as HDL, LDL, and certain smaller VLDL and remnants enter the intimal wall of arteries

where all except HDL adhere to elastin, which retards their exit and allows their accumulation (Figure 3). This concept would explain why chylomicrons are not considered atherogenic, despite severe hypocholesterolemia and why hypertension or hyperlipidemia, either individually or in combination, could exaggerate normal atherogenic processes (Krupp and Chalton, 1978).

A number of recent studies have documented a consistent negative correlation between plasma concentrations of HDL and clinically evident atherosclerosis. In its transit through the wall of the artery, HDL may incorporate esters into its central core for transport to excretory systems in the liver. The presumed role of HDL in clearing cholesterol from tissues may account for the observed increase in risk of atherosclerosis when HDL levels are relatively low, as in obesity, diabetes, and hyperlipidemic disorders and in physically inactive males.

Conversely, in chronic alcoholics, women in the childbearing age, and marathon runners, there is a high level of HDL. The "scavenger" role of HDL in clearing cholesterol from tissues could slow down atherogenesis and thereby contribute to the apparent protective effect of high plasma HDL in relation to atherosclerosis (Levy, 1978).

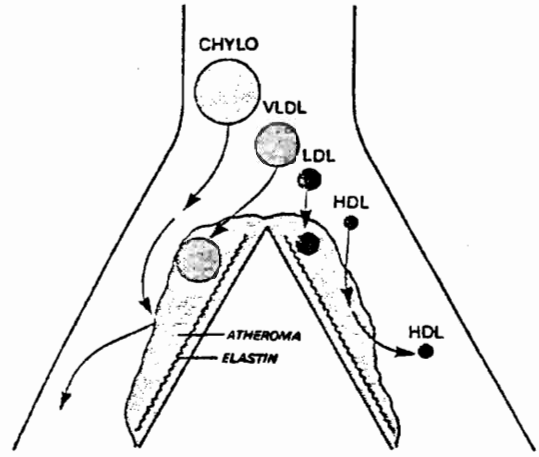

Figure 3. Lipoproteins and atheromas.

Note. From Current medical diagnosis and treatment by M. A. Krupp & M. J. Chatton. Los Altos: Lange Medical Publications, 1978. Reprinted by permission.

CHAPTER IV

CONCEPTUAL FRAMEWORK

In the early years of this century, doctors and nurses knew a great deal about making the best of an individuals' health if it proved to be inadequate for his needs and deteriorated from over-loading. Attitudes changed after the last war and people learned of the wonderful effects of new drugs such as antibiotics. Tuberculosis, for example, could be cured without the need for prolonged residence in a sanitarium; and pneumonia no longer required careful nursing and an anxious wait for the crisis to pass. Naturally enough, people sought this sort of cure for all their ailments and physicians, too, became more dependent upon drug treatments, more involved with physical and chemical body processes and less involved with the behavior and life styles of the patients.

Unfortunately for this trend in medicine, our behavior and circumstances cannot be put aside. Life style and fitness have a great deal to do with losing the ability to cope, becoming ill and breaking down. The chemical and physical processes inside the body are sensitive to the ways in which life is lived, but they have only a limited capacity for remaining normal.

It is, for example, quite natural for a man's blood pressure to be raised by prolonged periods of exhausting hard work and business preoccupations. If he does not train to become fit to

compensate for hard work, he may become the victim of self-induced chemical and hypertensive inflictions on the arteries. Attacks of illness from coronary disease are so common in Western urban life today, that they are often assumed to be determined by fate.

Being a social animal, man finds that many of the requirements for adaptation and for satisfying his needs stem from his relationships with his fellows. Unlike the ants and the bees, humans do not have a rigidly preordained role in society, but must continuously select among a vast array of options that offer abstract as well as concrete rewards and punishments.

Human beings are threatened by those very forces in society upon which they are dependent for nourishment, life and happiness. They must be part of the tribe, and yet are driven to give expression to their own proclivities. Because of this sensitive organization, they are often pulled apart two ways at the same time. Events having to do with their place in their society take on major significance. The individual is jeopardized not only by those forces that threaten survival of self and kin and opportunities for procreation, but also he is endangered when, through actions of other people, his growth, development and expression of individual proclivities are blocked (Wolff, 1950, p. 57). Man's lively appetite for challenge, exploration and for adventures, by driving him into situations frought with difficulty and hardships, may yield destructive arousal leading to ill-health and cardiovascular breakdown.

Executive health is a topic of concern particularly when one examines its correlation with cardiovascular disease. The Type A

behavior has already been shown to dominate in the executive population. They are remarkable for their warrior virtues. They do not recognize and give into fatigue like ordinary mortals. It is reasonable to postulate that the Type A's suffer more coronary disease by violating homeostasis by producing lipidemia and hypertension in their powerful reactions to everyday circumstances which threaten to get out of hand.

The body uses a variety of homeostasic devices to protect the constancy of the internal milieu against the changes which might be caused by our variable responses to the ever-changing circumstances in which we live. To be in a state of high arousal is to court danger from every point of view. Sooner or later homeostasis is violated and malfunctions ultimately capable of causing a breakdown are set in train because the consistency of the internal milieu cannot be maintained. The blood levels of cholesterol, triglycerides and uric acid tend to rise.

Some incredibly tough individuals can violate homeostasis and produce ill-health for years before they develop symptoms of impending cardiovascular breakdown. The sedentary executive living and working dangerously in an urban world of business hierarchy without an ongoing effort of keeping fit and maintaining healthy function may be inviting a self-destructive physical status. The individual who desires to maintain his own health learns not to jeopardize his freedom by violating his homeostasis, but trains himself to become fit enough and skillful enough to go forward as a health winner. The practice of assessing individuals for the factors which have been correlated with

a coronary breakdown in health--biochemical measurements and measurements of physical performance--is valuable in order to institute prophylactic programs in the prevention of manifestation of cardiac disease.

The essence of the male executive keeping well and avoiding a coronary breakdown is for each individual to learn to use his healthy function to a maximum, not fearing the initiative, persistence, leadership and hard work required for success, but utilizing vigilance and self-awareness to avoid self-defeating excesses of arousal. It is axiomatic that the body must be kept fit enough and tough enough for the demands of the mind.

It is obvious that we, as professionals, treating patients who are victims of destructive arousal, cannot relieve it by changing the world. However, education of these individuals in preparation for living with and reversing the destructive arousal is essential. An ongoing physical fitness program, as part of the executive's work week is imperative.

Operational Definitions

Cholesterol

A fatlike, pearly substance, a nonatomical alcohol, C_{27} H_{45} OH, crystallizing in the form of acicular crystals, and found in all animal fats and oils, bile, blood, milk, egg yolk etc. It constitutes a large part of the most frequently occurring type of gallstones and occurs in atheroma of the arteries, in various cysts, and in carcinomatous tissue (Doland, 1965).

Triglycerides

A circulating lipid. Free fatty acids derived mainly from adipose tissue are precursers of the endogenous triglycerides produced by the liver. Transport of endogenous triglycerides in association with beta-lipoproteins, the very low density lipoproteins (Krupp & Chatton, p. 1034, 1978).

Very Low Density Lipoproteins

(VLDL) the type of lipoprotein which contains high concentrations of triglycerides and moderate concentrations of both phospholipids and cholesterol (Guyton, 1976, p. 918).

Low Density Lipoproteins

(LDL) the type of lipoprotein which contain relatively few triglycerides but a very high percentage of cholesterol (Guyton, 1976, p. 918).

High Density Lipoprotein

(HDL) the type of lipoproteins which contain about 50% protein with smaller concentrations of lipids (Guyton, 1976, p. 918).

Good Level of Fitness

Measurement of Max VO_2 of $\geq$ 50 ml/kg/min on the Fisher-Fairbanks walking test.

Poor Level of Fitness

Measurement of Max VO_2 of $\leq$ 39 ml/kg/min on the Fisher-Fairbanks walking test.

Sedentary Office Workers

Telephone Company employees whose job description reads as follows:

Duties generally of an office and technically oriented nature.

1. Assigns daily workload for a central office maintenance forces.

2. Summarizes work content for special service orders, trunk orders, cable cuts, load valence cuts, and trouble reports to load into daily work.

3. Advises appropriate central office of pending customer trouble reports and acknowledges clearance of these troubles by central office personnel.

4. Works with central office switching control center supervisor, and central office foreman to record production results to determine future load and force forecasts.

5. Processing special service orders, cable make-up, prescription circuit design, etc. for special services engineer.

6. Preparation of cost studies.

7. Preparation of trunk facility charts and switching facility charts for switching design engineer.

8. Determines traffic capacities to be used for continuing administration of switching equipment components.

9. Analyzes data and schematic drawings related to central office equipment transitions of complex rearrangements.

CHAPTER V

DESIGN AND RESEARCH METHODOLOGY

Design

The research design was classified as descriptive (Brink & Wood, 1978). The variables measured in this study had been previously researched, and the technique for their measurements had been established with validity. The relationship among the variables was sought after the significance for studying these variables was related to a conceptual framework.

Fifty male desk workers, age 30 to 55, employed by Bell Telephone Company were utilized as the study sample population. All employees were excluded who had known cardiac disease, arrhythmias, angina, physical activity restrictions, known hypertension, pulmonary disease or an infectious process. This information was obtained by completion of a screening questionnaire (Appendix A) administered to interested employees at a prearranged meeting where details of the study were presented. Those employees interested in participating in the study, after the methodology was explained then signed a formal consent form and completed the checklist portion of the Minnesota Metabolic Activity Index Questionnaire (Appendix B). The researcher reviewed the health questionnaires in the order received and excluded any individual with a positive history for cardiac disease, arrhythmias, angina, pulmonary disease, known infections or activity

restriction. Of those eligible for the study, the researcher personally contacted each individual by phone to invite them to participate in the study. This process continued until a sample size of 50 was obtained. At the time of this phone conversation, appointments were made for collection of the physiological data at the YMCA facility in Salt Lake City.

Procedures

The research was conducted during the months of December, 1979, and January, 1980. The physical data was collected on each subject during scheduled appointments at the YMCA. Blood pressure, percent body fat, weight, HDL, LDL, VLDL, total cholesterol, triglycerides and Maximum VO_2 were the physical parameters measured. The Minnesota Metabolic Activity Index questionnaire/interview was administered to each candidate to quantify leisure time physical activities in kilocalories expended per day.

Source of the Data

This study proposed to examine the relationships of leisure time physical activity and lipids, VO_2, blood pressure and percent body fat in a group of homogenous sedentary male office workers. Therefore, the company physician of Bell Telephone Company was initially contacted and the proposed methodology was presented to him. From that meeting, arrangements were made to meet with the Personnel Specialist, who scheduled the researcher to meet at various times with small groups of male office workers aged 30-55. The details of the study were presented to this male population at these meetings.

Following the presentation, those men interested in participating in the study completed a health screening questionnaire (Appendix A), signed a consent form for participation in the study and completed the checklist section of the Minnesota Activity Metabolic Index Questionnaire.

The health screening questionnaires were then reviewed by this researcher. Any individual who answered questions positively for cardiac disease, arrhythmias, pulmonary disease, hypertension, hyperlipidemia, infections or activity limitations was disqualified from the study. Among the population, the first 50 who were free from the above, were selected to comprise the sample population. Each subject was then contacted by telephone, scheduled for an appointment at the YMCA at which time the physical parameters would be measured, and sent a follow-up letter (Appendix C). Additional subjects who qualified for the study were placed on a waiting list. Data from these alternates was collected if one of the initial 50 subjects was ill on the day of physical data collection or if a subject could not complete the fitness test. On three consecutive mornings, December 18, 19, 20, 1979, physical data was collected on 15 to 20 subjects each morning at the YMCA.

Population and Sample Selection

The objectives were to:

1. Secure a group of homogenous sedentary office desk workers.

2. Screen this population for a sample that was free from known cardiac disease, hypertension, hyperlipidemia, pulmonary

disease, infections or activity limitations.

3. Calculate AMI from data obtained from the subjects on the Minnesota AMI questionnaire/interview.

4. Calculate max VO_2 for each subject according to the Fisher-Fairbanks formula.

5. Calculate percent body fat for each subject.

6. Secure the services of a reputable laboratory for measuring a blood lipid profile on each subject.

Procedure for Collection of Data

The testing of the physiological parameters of blood pressure, skin-fold thickness for calculation of percent body fat, weight, blood samples for analysis of total cholesterol, HDL, VLDL, LDL and triglycerides and Maximum VO_2 was accomplished at the YMCA facility, 737 East 200 South in Salt Lake, Utah on 50 volunteer male subjects from Bell Telephone Company (Appendix D). All subjects arrived in a 12-hour fasting state. At the time of arrival each subject was questioned as to whether he was feeling well, and if he did desire to participate in the physiological data gathering. Each subject was given the opportunity to withdraw from the study and to ask questions which were answered by the researcher prior to the onset of the data collection procedure. Each subject was assigned a code number from 1 to 50 in the order in which he arrived to be used in the written account of the study and as a protection of confidentiality. Following is a description of the testing procedure used during a typical test morning.

Upon arrival at the YMCA facility, each subject sat quietly in

a chair and rested for 10 minutes. They were again instructed completely on the proceedings for all tests to be completed. This was done to avoid confusion and apprehension on the part of the subjects and to promote a smooth working testing operation. Following the resting period, each subject proceeded to Station I where the blood pressure was recorded. Blood pressure recordings were made by the Utah State Department of Health Hypertension Control Staff. The protocol is described in Appendix E. The subjects then proceeded to Station II. Here they were weighed on a standard platform balance beam scale and their weights recorded to the nearest pound. Each subject was dressed, but not wearing a jacket, coat or sweater, and pockets were emptied at the time of weight recording. Chest and axilla skinfold measurements were measured for calculation of percent body fat by an experienced technician. At the completion of these measurements the subjects proceeded to Station III.

At Station III, this researcher, trained in venipuncture, drew venous blood samples on all subjects. A vacutainer tube was labeled with the subject's name, current date and time specimen was drawn. The subject was asked to spell his name and state his name prior to the labeling of the tubes and drawing of the blood sample. The procedure for drawing the blood sample was supervised by the Director of the Chemistry Laboratory at LDS Hospital, Salt Lake City. The anticubital fossa area was cleansed with an alcohol swab in preparation for drawing a blood sample. A tourniquet was applied above the elbow and a blood sample drawn using a 20 g. disposable needle adapted to a 15 ml. vacutainer tube. After the blood sample

was obtained, the tourniquet was released and the wound covered with a sterile band-aid. At the conclusion of each morning's data collection, the researcher transported the samples to the LDS Hospital Chemistry Laboratory for determination of plasma lipid analysis according to the aca enzymatic method. The procedure used for lipid analysis is described in Appendix F.

The subjects then sat on a chair and rested for 10 minutes, then moved to the YMCA gymnasium where the Fisher-Fairbanks Fitness Test (Fairbanks, 1978) was administered by the Associate Director of Physical Education at the YMCA.

1. The exact distance of 100 feet on the testing area was clearly marked so that the rate of the walk could be measured accurately.

2. The subject rested for at least 10 minutes and then the therapist measured the number of apical pulse beats in 1 minute.

3. The test was begun by the participant walking in a normal walking pace so that the rate for 100 feet was between 15 and 16 seconds (4.26-4.55 mph or 6.82-7.28 km/hr). The pace was peridically monitored for consistency.

4. The time it takes to cover 100 feet was measured. The walking pace was adjusted so that the rate for 100 feet was between 15 and 16 seconds.

5. After walking for 5 minutes at a consistent pace, the participant came to a standstill and the therapist recorded the radial pulse rate immediately by starting the stopwatch on the first recovery heart beat and by counting the number of heart beats in exactly 10

seconds.

6. Maximum VO_2 can be estimated by placing the appropriate figures in the following regression equation: Max VO_2 (ml/kg/min) = 111.6 - 0.06190 x wt - 0.4564 x recovery heart rate - 0.0867 x Resting Heart rate. Weight is in pounds, recovery heart rate from 0-10 seconds is in beats per minute, and resting heart rate is in beats per minute. The standard error of estimation of this regression equation is $\pm$ 5.74 ml/kg/min and the percent error in predicting the mean max VO_2 is 11.9%.

7. Physical fitness levels can be categorized according to the following norms:

Max VO_2	Physical Fitness Category
below 29 ml/kg/min	Very poor
30-39 ml/kg/min	Poor
40-49 ml/kg/min	Average
50-59 ml/kg/min	Good
above 60 ml/kg/min	Excellent

There norms are similar to the norms accepted as valid by Cooper, 1968, p. 36. where he established fitness categories according to the number of minutes required to run 1.5 miles.

The Minnesota Leisure Time Activity Questionnaire (LTA) was studied in a group of 175 men derived from a Twin Cities population (Taylor et al., 1978). It is an interview-administered questionnaire (Appendix B) to evaluate energy expenditure in leisure physical activity. The Minnesota LTA Questionnaire has been validated in several studies. It is currently used in Multiple Risk Factor

Intervention Trial, a collaborative prevention trial, sponsored by the National Heart, Lung and Blood Institute. The LTA is also currently utilized in an ongoing study of 10,000 Minneapolis-St. Paul residents which will continue over the next 5 years (Jacobs, 1980).

Energy expenditure is expressed as scores for an Activity Metabolic Index i.e., Total AMI, Light AMI, Moderate AMI, and Heavy AMI. The Total AMI score is in correlation with total kilocalories expended on leisure time physical activity.

Prior to administering the Interview portion of the Minnesota Metabolic Activity Index, the manual for administration was obtained from Joan M. Knudsen, Community Programs Specialist, School of Public Hygiene, University of Minnesota, who was trained by Drs. Jacobs and Taylor at the Health Sciences Center in the administration of the interview portion. The researcher reviewed the interviewing technique as described in detail in the manual, then taped two pilot interviews and critiqued the tapes for gaining rapport with the interview subject, using clarifying probes, avoiding loaded questions and for using a standard time-frame to complete the interview. The researcher then consulted Dr. Jacobs at the University of Minnesota by telephone for further advice regarding administration of the interview. Each subject was scheduled for an interview by the researcher, lasting from 30 to 40 minutes. Following completion of these interviews, the scores were computed for Total AMI, Heavy AMI, Moderate AMI and Light AMI.

Instruments

Instruments were required to measure blood pressure, skin-fold

thickness for calculation of percent body fat, Activity Metabolic Index and Max VO_2.

Blood Pressure

Blood pressures were recorded by the Hypertension Control Staff of the Utah State Department of Health, which consisted of three registered nurses. The staff had been standardized in blood pressure measurements as described in Appendix E, without significant variance.

Skin-Fold Thickness for Calculation of Percent Body Fat

An individual's body composition can be most accurately estimated by the underwater weighing technique (Pollock, 1978). Although quite accurate, determining body fat by the underwater weighing technique is not very practical. Therefore, anthropometric measurements were used to estimate the various components of body composition, that is, body density and relative or absolute fat. The equipment was inexpensive, required little or no space, and the measurements could be quickly and easily obtained. Therefore skin-fold thickness measurements could be used rather efficiently in testing this sample population ($N = 50$).

The Harpinden calipers were used to obtain chest and axilla skin-fold measurements using the technique of Behnke and Wilmore (1974). The skin-fold was grasped firmly by the thumb and index finger, and the caliper was placed on the exact site approximately one-fourth inch from the thumb and finger. The chest skin-fold was measured as the diagonal fold one-half of the distance between the

anterior-axillary line and nipple. Axilla skin-fold was measured as the vertical fold on the midaxillary line at approximately the line of the nipple (xiphoid process at lower end of the breast bone). Previously determined reliability coefficients for the technician were 0.96 and 0.97 for the axilla and chest skin-folds respectively. Density of body fat was calculated according to the formula of Pollock developed at the Institute for Aeorbic Research at the University of Houston: Density = 1.0766 - 0.0098 x chest skin-fold - 0.0053 x axilla skin-fold. To convert density to percent body fat the Siri percent formula was utilized: Percent Fat = 4.9501/Density - 4.50 x 100 (Pollack, 1978).

Measurement of Maximum VO_2

Maximum oxygen uptake (aerobic capacity) is the largest amount of oxygen that one can utilize under the most strenuous exercise (Pollock, 1978). Because maximum oxygen uptake generally summarizes what is going on in the oxygen transport system (including cellular utilization) during maximum or exhaustive exercise, and can be measured rather easily, Max VO_2 has been used as the measure most representative of cardiorespiratory fitness. A larger person generally has more muscle mass, and thus the capability of burning more oxygen; aerobic capacity is expressed in milliliters of oxygen per kilogram of body weight per minute (ml/kg/min).

Difficulty arises in setting a standard for optimal fitness because a specific line of aerobic capacity for optimal health has not been determined. Sedentary middle-aged males characteristically fall below 40 milliliters per kilogram of body weight per minute of

oxygen uptake. This value drops to 30 by age 50 to 60. Endurance runners characteristically are able to increase their Max VO_2 to $\pm$ 75 milliliters per kilogram per minute of oxygen uptake (Pollock, 1978, p. 36).

The oxygen uptake increases during the first minutes of exercise to a "steady state," where the oxygen uptake corresponds to the demands of the tissues. When the exercise stops, the oxygen uptake gradually decreases to the resting rate; the oxygen debt is paid off. From a methodological viewpoint, it is important to emphasize that maximum oxygen uptake is obtained at a workload that is not necessarily maximal. An all-out test is not necessary for the assessment of an individual's maximum aerobic power. In studies where maximal oxygen uptake is to be measured, the collection of expired air or other measurements may start after about 1 minute of exercise, provided that the work load is extremely heavy (super-maximal) and is preceded by a warming-up period, but it is wise to aim at a work period of 5 minutes. In repeated determinations of maximal oxygen uptake on the same subject, the standard deviation is 3%, which includes biological and methodological variables (Astrand & Rodahl, 1977).

The classical laboratory studies, with subjects working continuously for 5 minutes or longer provide standardized conditions and permit comparisons to be made on repeated occasions. When the work time is extended to about 1 hour, the oxygen uptake, heart rate, and cardiac output are maintained at the same rate as attained after about 5 minutes of exercise, provided the oxygen uptake is not

higher than about 50% of the maximum (Astrand, Astrand, & Rodahl, 1959, p. 302).

In laboratory experiments, three methods of producing standard work loads have been mainly applied: running on a treadmill, working on a bicycle ergometer and using a step test. The work should involve large muscle groups, and the measurements of the O_2 uptake should be started when the work has lasted a few minutes, to allow the oxygen uptake to reach its maximum.

The critical question is whether or not the different types of work mentioned give the same maximal oxygen uptake. A number of studies have been undertaken to clarify this question. Running on the treadmill uphill ($\geq$ 3° inclination), may bring the O_2 uptake to a maximum whereas running horizontally or at a slight inclination may result in a somewhat lower maximal oxygen uptake (Taylor, Buskirk, & Henschl, 1955). Bicycling produces, on the average, a lower oxygen uptake, at least compared with running uphill. In studies in which objective criteria have been used to determine whether the maximal oxygen uptake had been reached for the type of work in question, the values for running are on an average 5 to 8% higher than for bicycling (Astrand & Rodahl, 1978, p. 335).

The standard error of the method for the prediction of maximal oxygen uptake from submaximal exercise tests is about 10% in relatively well-trained individuals of the same age, but up to 15% in moderately trained individuals of different ages (Astrand, 1960).

During bicycling, the subject may often experience a feeling of local fatigue or a sensation of pain in the thighs or knees. The

discomfort may cause the work effort to be interrupted before the oxygen-transporting organs have been fully taxed. In persons who have never ridden a bicycle, a maximal test on the bicycle may be undesirable as a method to assess oxygen uptake.

Disadvantages arise with the treadmill exercise modality as a means of measuring maximum VO_2. The treadmill is expensive and immobile. Older individuals may have some difficulty in walking on the treadmill. The provision of a handrail for support will make the work load still more unpredictable. Changing of speed on the treadmill can be a disadvantage. There will be a speed at which some subjects prefer to walk (e.g. with long legs) and others will run. The energy demand will be different and cannot be predicted from speed alone. The step test has a more limited application, due to poor standardization and offering limited provisions for varying the load on the oxygen-transporting system.

The classical method for the determination of oxygen uptake, the Douglas bag method, rests on a very secure foundation. Theoretically sound, the bag method has been tested under a wide variety of circumstances and is unsurpassable in accuracy.

A disadvantage with the method is, again the expense and the fact that the subject is somewhat hampered by the equipment required for collection of expired air. This limits the subjects' freedom of movement. Furthermore, the analysis merely provides a mean figure for the oxygen uptake depending upon the length of time in which the expired air is collected.

In the clinical examination of patients or presumably healthy individuals determination of functional capacity via an exercise test is recommended, particularly prior to that individual beginning a physical fitness program. The proposed idea of everyone visiting their physician's office to obtain a sophisticated exercise test is impractical and cost ineffective. Mass public evaluation for aerobic capacity could be implemented if the participant is tested safely and properly, with an end-result of a good estimate of functional capacity. Such mass evaluation must involve an efficient and inexpensive means of testing.

Regression equation formulas are available for the calculation of energy cost of walking (Van der Walt & Wyndham, 1973) and the simplest and most extensive applied method of testing functional capacity is to determine heart rate during or immediately after exercise (Astrand & Rodahl, 1977, p. 344).

Based on these principles, the Fisher-Fairbanks Walking Test (Fisher, 1978) was developed by the Human Performance Research Center, Brigham Young University, Provo, Utah. Maximum VO_2 can be estimated by placing the appropriate figures in the following developed regression equation: Max VO_2 (ml/kg/min) - 111.6 - 0.06190 x wt - 0.4564 x recovery heart rate - 0.0867 x resting heart rate. The standard error of estimation of this regression equation is $\pm$ 5.74 ml/kg/min and the percent error in predicting the mean maximal VO_2 is 11.9% (Fairbanks, 1978).

The efficiency and convenience of utilizing the Fisher-Fairbanks test as a method of measuring VO_2 supercedes other VO_2

measurement methods discussed for use in a large sample population of testing.

The Minnesota Leisure Time Activity Questionnaire

Energy expended in a specific activity is estimated as the product of the intensity Code I and the duration of exercise in minutes for a year D. The ratio of metabolic rate during work to the basal metabolic rate provides an intensity code. The Activity Metabolic Index is expressed as AMI = I x D. The intensity codes are based on experimentation in which rates of oxygen consumption (VO_2) were measured while people performed various specific activities (Passmore & Durnin, 1955; McDonald, 1961). For many activities, VO_2 is a function of repetitions of an activity done at a constant rate (shoveling). A consistency was found in the intensity codes used in the Minnesota LTA and those employed by Reiff and Montage (1967).

The Minnesota AMI assessment is a questionnaire-interview form. The respondent partially fills out the form alone and the interviewer fills out detailed information for each activity checked. The specific months and their number (M) in which the activity was performed, the average number of occasions in each such month (F) and the average duration of activity on each such occasion (T) are all queried. The overall AMI is: Total AMI = $\Sigma(I \times M \times F \times T)$.

To categorize men in this manner with the Minnesota LTA scoring, Light AMI is defined by summing only those activities having intensity codes 2.0, 2.5, 3.0, 3.5, and 4.0. Moderate AMI is obtained by summing over activities with intensity codes of 4.5, 5.0, and 5.5

Heavy AMI is defined by summing over all activities having intensity codes $\geq$ 6.0. The Total AMI = Light AMI + Moderate AMI + Heavy AMI.

Delimitations

The experiment was delimited to:

1. Clerk workers at Bell Telephone Company.
2. Male subjects only.
3. Subjects whose ages range from 30-55.
4. Subjects who were free from known coronary disease, hypertension, hyperlipidemia, pulmonary disease, infections, activity restrictions.

Limitations

The experiment may have been limited because:

1. No more than 50 subjects were included due to the cost of testing.
2. The data was collected between Thanksgiving and Christmas when due to tradition caloric intake may have been greater.
3. The season of the year (winter) may, by itself, have had some effect on the amount of leisure-time activities.
4. The calculation of the Minnesota Activity Metabolic Activity Indexes may have been subject to a margin of inaccuracy due to the interviewer's lack of previous experience in administering the interview portion of the questionnaire.

Protection of Human Subjects

All members collecting data were trained in venipuncture technique and cardiopulmonary resuscitation. The population was

screened to determine a sample of subjects free from known cardiac disease or hypertension. All questions asked by the subjects were answered prior to their participation in the study. The subjects were free to withdraw from the study at any time and were dismissed on the day of data collection if they were not feeling well.

The subject's confidentiality was protected. The data collected was assigned a code number and only this code number was referred to in the written account of the study. At no time was the subject referred to by name. A copy of the lab work and fitness evaluation outcome was sent to the subject and to his personal physician, if the subject so requested (Appendix H).

CHAPTER VI

RESULTS

The range, mean and standard deviations of the variables are recorded in Table 1 and Table 2. Pearson product correlation coefficients among AMI, blood pressure, body fat and weight are given in Table 3. Maximum VO_2 was correlated with blood pressure, lipids, body fat and weight in Table 4.

A multivariate analysis was performed with the group as the dependent variable and VO_2, HDL, LDL, VLDL, cholesterol, triglycerides and percent body fat as the independent variables.

Statistics

Pearson correlation coefficients were determined to establish a relationship between the variables and a measurement of strength of the variables. Multivariance analysis was calculated to see relationships between groups.

Table 1

Variables Mean and Standard Deviation

($\underline{N}$ = 50)

Variable	Minimum	Maximum	Mean ± SD
VO_2	19.43	50.88	33.22 ± 6.23
Systolic Blood Pressure (mm Hg)	108.00	158.00	129.28 ± 11.31
Diastolic Blood Pressure (mm Hg)	60.00	98.00	80.00 ± 8.14
Total Cholesterol (mg/DL)	120.00	315.00	209.44 ± 38.35
LDL Cholesterol (mg/DL)	59.00	199.00	133.96 ± 29.70
HDL Cholesterol (mg/DL)	24.00	76.00	43.96 ± 10.95
VLDL Cholesterol (mg/DL)	11.00	62.00	27.31 ± 13.61
Triglycerides (mg/DL)	54.00	698.00	163.02 ± 126.38
Percent Body Fat (%)	12.18	24.59	18.00 ± 3.27
Weight (lbs)	128.50	256.00	184.50 ± 27.66

Table 2

Means and Standard Deviations of Activity Index Scores

Variable	Minimum	Maximum	Mean ± SD
Total AMI (Kcal/D)	34.19	1109.07	296.63 ± 187.94
Heavy AMI (Kcal/D)	18.41	856.11	137.71 ± 138.72
Moderate AMI (Kcal/D)	8.7	397.00	84.77 ± 71.54
Light AMI (Kcal/D)	0.0	313.00	64.78 ± 59.61

Table 3

Correlation Matrix of AMI with Blood Pressure,

Lipid Variables, Body Fat and Weight ($\underline{N}$ = 50)

	Max VO_2	Systolic B/P	Diastolic B/P	Cholesterol	LDL	HDL	VLDL	Triglycerides	Body Fat	Weight
Total AMI	.42	-.13	-.33	-.41	-.33	.20	-.32	-.22	-.36	-.22
Heavy AMI	.39	-.06	-.16	-.33	-.24	.15	-.29	-.14	-.17	-.10
Moderate AMI	.16	-.21	-.28	-.30	-.30	-.29	-.33	-.22	-.27	-.26
					$\underline{P} \leq .05$					
Light AMI	.11	.02	-.07	-.16	-.10	-.13	.05	-.02	-.27	-.11
	$\underline{P}$ = .21	$\underline{P}$ = .44	$\underline{P}$ = .29	$\underline{P}$ = .13	$\underline{P}$ = .24	$\underline{P}$ = .19	$\underline{P}$ = .38	$\underline{P}$ = .45	$\underline{P}$ = .02	$\underline{P}$ = .22

Table 4

Correlation Matrix of VO_2 and Blood Pressure, Lipids, Body Fat and Weight ($\underline{N}$ = 50)

	Systolic B/P	Diastolic B/P	Cholesterol	LDL	HDL	VLDL	Triglycerides	% Body Fat	Weight
VO_2	-.02	-.13	-.31	-.40	.33	-.26	-.27	-.30	-.09
Systolic		.53	.23	.30	-.03	.09	.32	.25	.29
Diastolic			.32	.23	-.26	.40	.33	.34	.21
Cholesterol				.90	.14	.40	.36	.40	.20
LDL					-.04	.16	.15	.28	.17
HDL							.98	-.16	-.22
VLDL							.30	.21	.23
Triglycerides								.26	.15
% Body Fat									.70

$\underline{P}$ = .05

CHAPTER VII

FINDINGS AND ANALYSIS

VO_2 was significantly positively correlated with HDL (r = .33) and negatively correlated with VLDL (r = -.26), negatively correlated with triglycerides (r = -.27) negatively correlated with cholesterol (r = -.31), negatively correlated with percent body fat (r = -.30) but was not significant with body weight (r = .09), or systolic pressure (r = -.02) or diastolic pressure (r = -.13). The total AMI demonstrated a modest positive association with VO_2 (r = .42), a modest negative association with VLDL (r = -.32), triglycerides (r = .22). HDL showed a small but negative correlation with percent body fat (r = -.16) and weight (r = -.22), as well as with diastolic pressure (r = .26). There was a higher correlation between Heavy AMI and VO_2 (r = .39) than with moderate AMI and VO_2 (r = .16) or with Light AMI and VO_2 (r = .11). Statistically, none of the correlations with light AMI were significant. Diastolic pressure and Maximum VO_2 showed a small negative correlation (r = -.13); a stronger negative correlation existed between Total AMI and distolic pressure (r = -.33). There was no significant correlation with systolic blood pressure.

These results were similar to the Pearson correlation coefficients obtained by Schwane and Cundiff (1979) who researched the relationships between physical activity and plasma lipids and between cardiorespiratory fitness and plasma lipids in 152 young adults

(Table 5). An exception to the likeness in the statistical results of these two studies is that this study demonstrated a positive correlation (r = .40) between total cholesterol and body fat, whereas the study by Schwane and Cundiff revealed an insignificant correlation (r = -.06). However, the overall mean cholesterol in their study was 145.5 ml/dl, compared to the mean cholesterol value in this study of 209.44 mg/dl. The significantly lower mean cholesterol value would perhaps account for the insignificant correlation authors Schwane and Cundiff obtained.

The sample population of sedentary male office workers overall demonstrated low levels of fitness (Table 6) only one subject who jogged 45 minutes three times per week fell into the good fitness category. Ninety-two percent of the sample were in the poor or very poor fitness level. Six percent were of average fitness and 2% of good fitness.

The range for Total AMI scores was 34.19-1109.07 with SD ± 184.94 (Table 7). The range for Heavy AMI (Table 8) scores was 18.41 - 856.11 with SD ± 138.72. The range for Moderate AMI was 8.7 to 397 with SD ± 71.54 (Table 9). The range for Light AMI was 0 to 313 with SD ± 59.61 (Table 10). A broad range in scores was also noted with triglycerides (54-698 with SD ± 126.38 (Table 11).

The standard deviations can be accounted for by the broad range of scores in these variables and the sample population size (N = 50).

In the multivariate analysis (Table 12) knowledge of cholesterol, VLDL, diastolic pressure or Total AMI added no predictability

Table 5

Comparison of Pearson Correlation Coefficients among the Variables of this Study and a Similar Study by Schwane and Cundiff, 1979

	Schwane & Cundiff	Present Study
Treadmill with HDL	.29	(VO_2 with HDL) .33
Treadmill with LDL	-.06	(VO_2 with HDL) -.14
Treadmill with Triglycerides	-.19	(VO_2 with Trigly) -.27
HDL with Body Weight	.21	.23
HDL with Percent Body Fat	.18	.21
LDL with Percent Body Fat	.11	.27
Choesterol with Body Fat	-.06	.40
Triglycerides with Body Fat	.18	.26
Cumulative Aerobic Points with HDL	.14	Total AMI with HDL .20
Treadmill with Body Fat	-.65	(VO_2 with Body Fat) -.35

Table 6

Maximum VO_2 Scores and Corresponding Fitness Category Placement ($\underline{N}$ = 50)

Fitness Category	VO_2 Range	$\underline{N}$ =
Very Poor	below 29 ml/kg/min	10
Poor	30-39 ml/kg/min	36
Average	40-49 ml/kg/min	3
Good	50-59 ml/kg/min	1

Table 7

Scatterplot of Total AMI Scores

and Physical Fitness Category

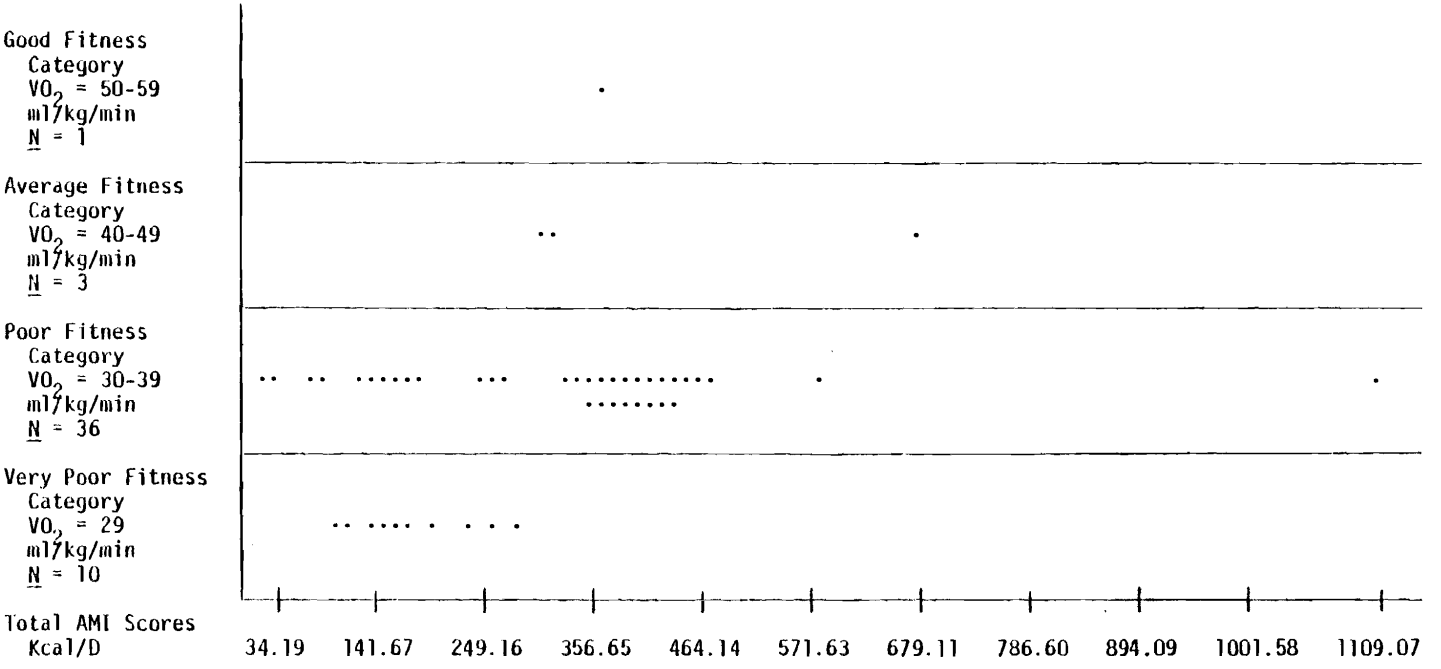

Table 8

Scatterplot of Heavy AMI Scores

and Physical Fitness Category

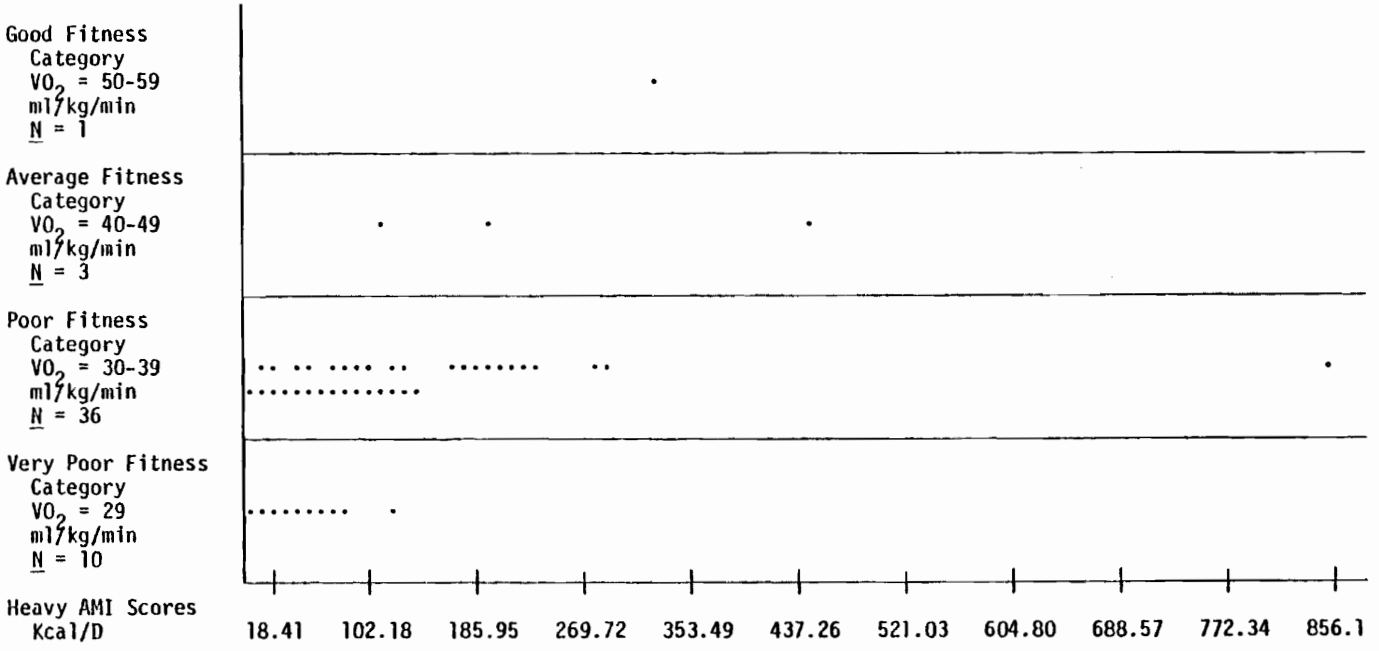

Table 9

Scatterplot of Moderate AMI Scores

and Physical Fitness Category

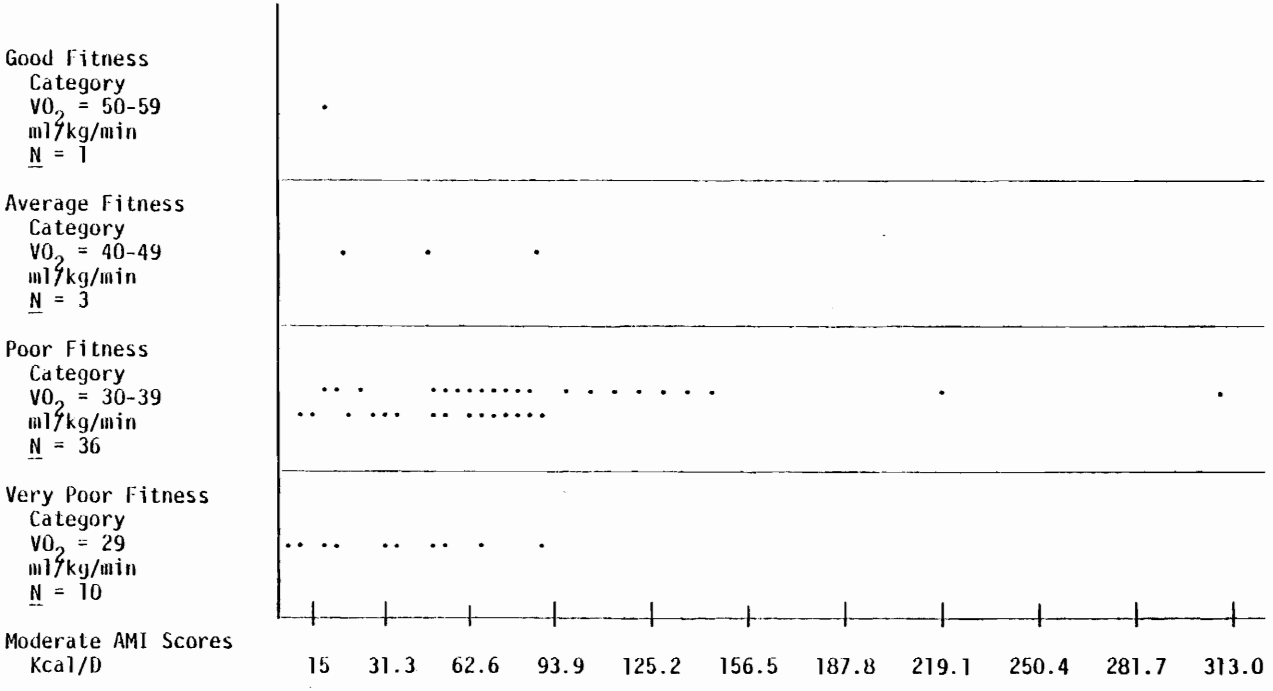

Table 10

Scatterplot of Light AMI Scores

and Physical Fitness Category

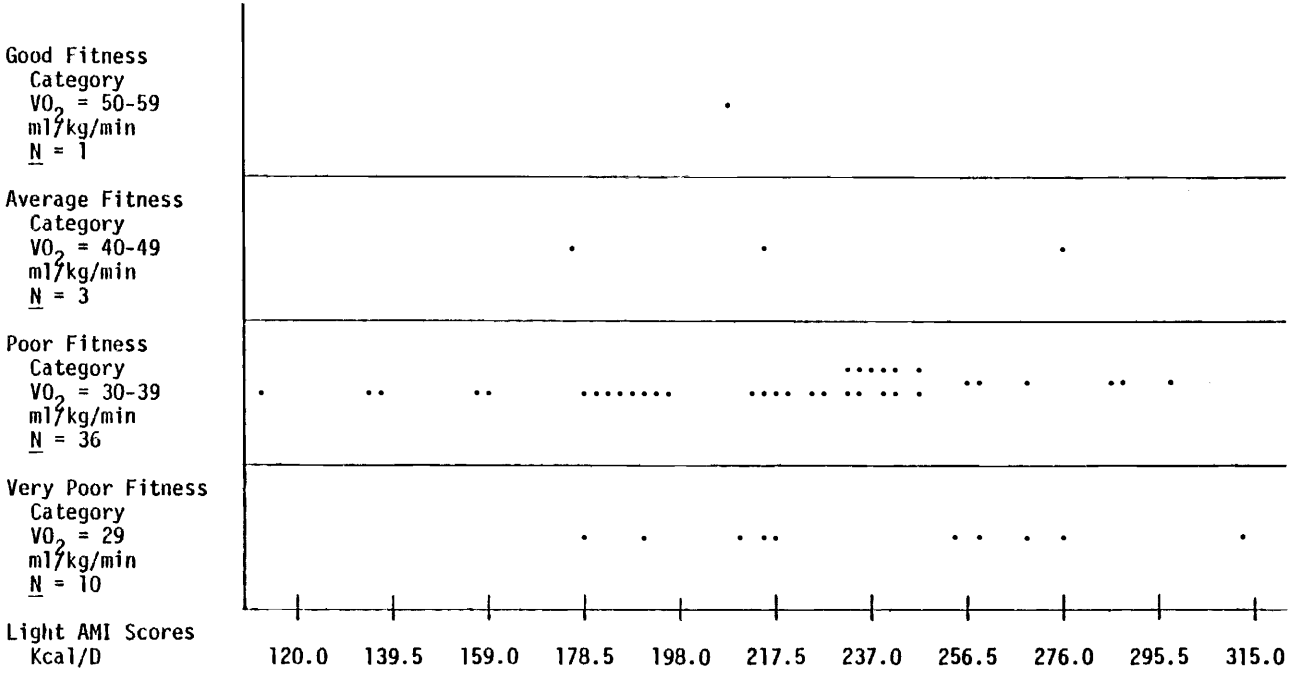

Table 11

Scatterplot of Triglycerides and Physical

Fitness Category

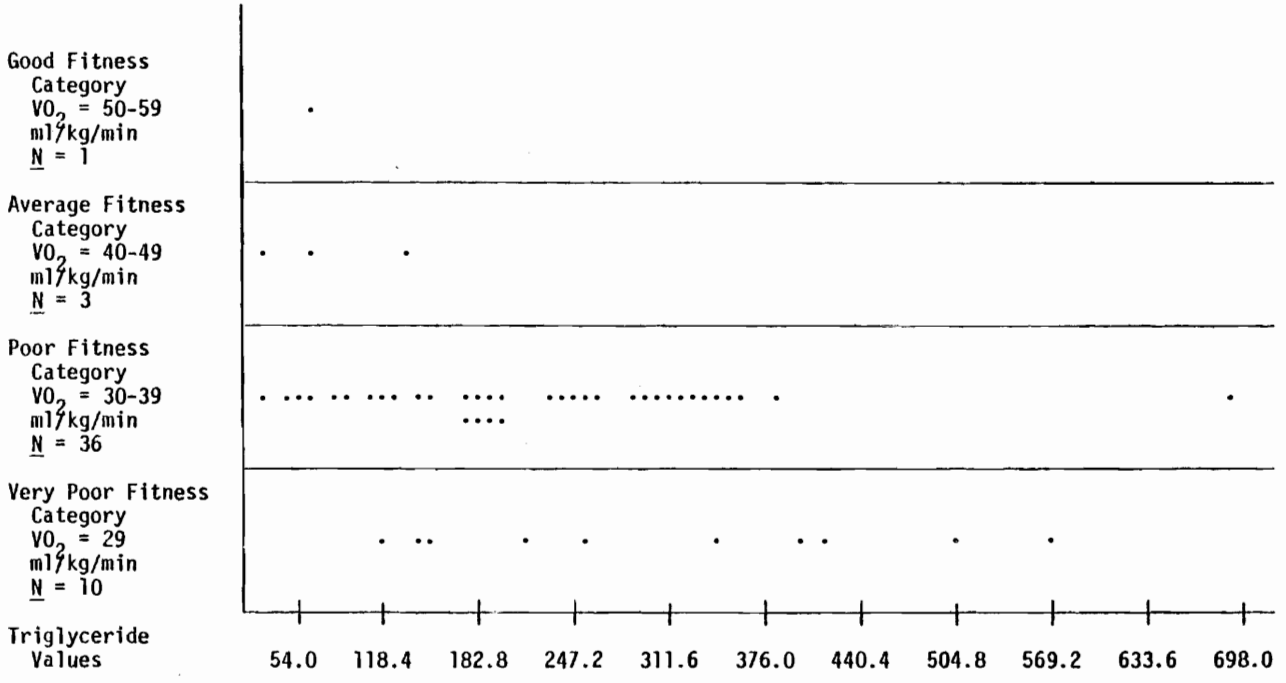

Table 12

Multiple Regression Coefficient (R) Between Group (Dependent Variable) and VO_2, HDL, LDL, VLDL, Cholesterol, Diastolic Pressure and Total AMI

Dependent Variable	Independent Variables (Correlation)				
Group	VO_2				
	Analysis of variance	df			
	Regression	1	R = 0.79	F = 174.34	P = .0001
	Residual	45			
	HDL				
	Analysis of Variance	df			
	Regression	2	R = 0.84	F = 119.92	P = .0001
	Residual	44			
	LDL				
	Analysis of Variance	df			
	Regression	3	R = 0.85	F = 84.81	P = .0001
	Residual	43			
	Cholesterol				
	Analysis of variance	df			
	Regression	4	R = 0.86	F = 67.13	P = .0001
	Residual	42			
	VLDL				
	Analysis of variance	df			
	Regression	5	R = 0.97	F = 308.83	P = .001
	Residual	41			
	Diastolic Pressure				
	Analysis of variance	df			
	Regression	6	R = 1.50	F = 4.53	P = .001
	Residual	40			
	Total AMI				
	Analysis of variance	df			
	Regression	8	R = 1.60	F = 2.20	P = .001
	Residual	38			

beyond VO_2, HDL and LDL (r = 0.85).

Hypotheses (Testing)

1. There will be a positive correlation between Total Activity Metabolic Index (AMI) and HDL and a negative correlation between AMI and cholesterol, LDL, VLDL and Triglycerides.

Pearson correlation coefficients demonstrated a positive correlation of AMI and HDL (r = .20) and negative correlation between AMI and LDL (r = -.33), VLDL (r = -.32) triglycerides (r = -.20) and cholesterol (r = -.41). On the basis of these statistical tests, the hypothesis was accepted.

2. There will be a positive correlation between percent body fat and cholesterol, LDL, VLDL and triglycerides, and a negative correlation between percent body fat and HDL.

Pearson correlation coefficients demonstrated a positive correlation with body fat and cholesterol (r = .40) with LDL (r = .28), with VLDL (r = .21), with triglycerides (r = .26) and a negative correlation with HDL (r = -.16). The hypothesis was accepted.

3. Subjects with higher Total AMI scores will have higher VO_2 values. The correlation between VO_2 and Total AMI was r = .42. The hypothesis was accepted.

4. Subjects with higher VO_2 scores will demonstrate higher HDL. The correlation between VO_2 and HDL was r = .33. The hypothesis was accepted.

5. Subjects with higher VO_2 scores will demonstrate lower LDL. The correlation between VO_2 and LDL was r = -.14. On the basis of the small correlation, the hypothesis was rejected.

6. Those subjects with higher VO_2 scores will demonstrate lower triglycerides and cholesterol.

The correlation between VO_2 and triglycerides was $\underline{r}$ = -.20 and with cholesterol was $\underline{r}$ = -.33.

The hypothesis was accepted.

CHAPTER VIII

DISCUSSION

Total Activity Metabolic Index (Kilocalories/day) and Max VO_2 showed a small, but significant correlation, in terms of the sample size, with HDL lipoprotein (Total AMI with HDL $\underline{r}$ = .20 and Max VO_2 with HDL $\underline{r}$ = .33). The group was regarded as relatively sedentary and homogeneous in on-the-job activity and leisure time activities. There was only one subject who jogged regularly (45 min. three times/week) and he correspondingly demonstrated a higher HDL score (76 mg/DL).

Previous studies have demonstrated the higher HDL levels in elite runners (Wood & Haskell, 1979) and an increase in HDL in a control group exercised regularly for as short of time as one week (Erkelens, 1978). This cross-sectional studied demonstrated that HDL levels remained below reported atherogenic-protective levels in a group of sedentary male office workers whose mean leisure time Total Metabolic Activity Index Score was 296.63 K/cal/day and VO_2 of 33.22 ml/kg/min (poor level of fitness category). Pollock (1973) found that men who participate in activities having intensity codes of $\geq$ 6.0 will have larger aerobic capacities than men who participate in activities having intensity codes < 6.0. This current study also suggested a higher correlation between Heavy AMI and VO_2 ($\underline{r}$ = .39) than aerobic capacity with moderate AMI ($\underline{r}$ = .16) or with light AMI ($\underline{r}$ = .11) The association of the heavy activity score with treadmill performance is

consistent with physiological theory and experience. On the other hand, the association does not constitute a complete validation of the instrument. Possibly, high level leisure time physical activities are reported with more accuracy than moderate or low activities. Additional reproducibility studies are needed.

Body composition may reflect to an extent, level of regular physical activity or fitness, since caloric expenditure is a determinant of fat deposition in or removal from adipose tissue. Max VO_2 showed a negative correlation with percent body fat ($\underline{r}$ = -.35).

Limitations of the indicators of leisure time physical activity and aerobic fitness used in this study must be recognized. Intuitively, low validity and reliability must be expected for activities other than walking, jogging and cycling, i.e. activities that vary greatly in intensity and/or whose energy expenditure varies depending on efficiency of performance. The interviewing process proved to be most tedious. The researcher found difficulty in maintaining control of the interviewing process. The subjects frequently requested counseling for their particular health situation, or would not answer in short phrases, but rather tended to elaborate on activities. As the researcher gained more experience with the process, the interviewing process was reduced. A minimum of 30 to 40 minutes was required for each interview, and some interviews required up to an hour's time. The time-factor involved for interviewing was far beyond that anticipated by this researcher. Therefore, the actual time required for the interviewing process needs to be considered for replication of this study.

With regard to the Fisher-Fairbanks walking test as a measurement of VO_2, all of the participants, except for the jogger, slowed their pace of performance during the last minute of the exercise. The calibrated pace was 100 feet per 15 seconds whereas a recheck calibration revealed the pace to be 100 feet covered over 20 to 25 seconds during the final minute of the total 5 minute exercise period. Additional research by Fisher (1980) has demonstrated the resting heart rate to vary day to day in study subjects by as much as 9 beats per minute. Therefore, another regression equation for estimation of VO_2 from walking is currently being computed using age rather than heart rate.

Overall correlation of max VO_2 and AMI suggested a stronger relationship between total AMI and VO_2 ($\underline{r}$ = .42) than with Heavy AMI ($\underline{r}$ = .16) or with Light AMI ($\underline{r}$ = .11). However, scrutiny at individual subject's values revealed that the subject with the highest VO_2 (50.88 me/Kg/min) did not have a correspondingly higher AMI score. Closer examination was made with regard to VO_2 and AMI scores on subjects who fell into good and average physical fitness categories. The subject with a max VO_2 value of 50.88 ml/Kg/min was calculated to have a total AMI score of 300.82 K/cal/D and a Heavy AMI score of 228.92 Kcal/D. However, his Heavy AMI score was comprised mainly of the numbers calculated for jogging 45 minutes three times per week the year round. Other subjects accumulated higher AMI scores (i.e., subject 34, Total AMI = Kcal/D, with a VO_2 of 35.26, Subject 38 Total AMI 475.66 with a VO_2 of 36.56) with lower VO_2 values. Subject 34 accumulated Heavy AMI points (intensity of

activity $\geq$ 6.0) through activities of playing basketball for three hours/week, four months out of the year and swimming four times per year for 30 minutes duration. Subject 38 achieved a total AMI score of 475.46 with a VO_2 of 36.56 ml/kg/min. His Heavy AMI was calculated from his stated activities of mountain climbing for a 21 day period, three hours one day, water skiing three times per year for 25 minutes, swimming at the beach three times per year for 30 minutes, playing a basketball game four times per month yearly for one hour duration, and snow shoveling four times per month for four months for 30 minutes each.

Therefore, the type of activity which accounted for scores calculated must be examined. From this break-down of activities, it was evident that a regularly weekly aerobic activity (although earning a lower score in kcal/D expenditure) resulted in a higher VO_2 score.

Various activities of high intensity may be accumulated throughout the year, but if they were not carried out on a regular basis, a lower VO_2 value was measured.

Summary

In summary, within this homogeneous and sedentary group of male office workers, who demonstrated relatively sedentary leisure time activities, there was a small but statistically significant relationship with outside leisure activity, aerobic fitness and plasma lipids; HDL increasing with greater amounts Kcal/D energy expenditure. The fact that the difference in outside leisure time

physical activity was small among the subjects investigated may account for a positive but small numerical correlation. These relationships suggest that outside leisure time activity with a mean of 296.63 Kcal/D is not a vigorous enough activity to increase aerobic fitness or to increase the HDL to an athrogenic-protective level. Levels reported as atherogenic protective in the literature were 56-75 mg/DL (Wood & Haskell, 1979) whereas the mean HDL level in this study was 43.96 mg/DL.

A number of aspects of this investigation indicate the need for further research. The extent to which certain physiological concomitants of the active lifestyle, rather than exercise per se, may account for the lipoprotein differences remains to be determined by experiments designed to distinguish these factors. Leanness is the chief potential candidate here. The mechanism by which increased exercise (or its accompanying changes) leads to the characteristic lipoprotein pattern is not definitely known. The important general question remains unanswered: would a change from the lipoprotein pattern of these male office workers studied, to that of the typical chronic exerciser (however achieved) result in a worthwhile reduction of atherosclerotic disease?

From this study, it can be concluded that a cross-sectional investigation of American sedentary office workers reveals a marked lack of outside leisure time physical activity and resulting higher risk profile for development of atherosclerosis. Therefore additional research is necessary to determine the cost-effectiveness and physiological benefits of an occupational based and professionally

directed on-going fitness program for subjects such as were studied in this investigation.

APPENDIX A

QUESTIONNAIRE

Please Print Legibly

Name _____

Home Address _____

_____ Zip Code _____

Home Phone _____

Job Position at Bell Telephone _____

How can you be reached at Bell Telephone _____

Age _____ Weight _____ Height _____

If you qualify for the study and agree to participate, a copy of the tests results will be sent to you at your home address. Would you also like to have a copy mailed to your personal physician?

If yes, Physician's Name _____

Address _____

Check in front of those questions to which your answer is YES. Please provide the additional information requested for the YES answers. Leave the others blank.

____ 1. Has a doctor ever diagnosed you as having high or low blood pressure? Record you blood pressure the last time it was taken, if known. _____

____ 2. Have you ever had a heart attack? When _____

____ 3. Do you currently have pain in your heart or chest (angina)? How often do you get these chest pains? _____
What brings them on? _____

Are you needing to take nitroglycerin tablets to relieve the chest pains? _____

Do the chest pains occur at rest or only with physical activity? _____

____ 4. Do you have a heart murmur? _____

____ 5. Do you ever notice extra heart beats or skipped heart beats? Has the doctor ever told you that you have an irregular heart rhythm? _____

Are you currently being treated by your doctor for an irregular heart rhythm? _____

____ 6. Has a doctor ever told you that you have any type of heart disorder? _____

____ 7. Has a doctor ever told you that you have an abnormal electrocardiogram? (Date and abnormality, if know) _____

_____

____ 8. Do you have any lung disorders? (If so, explain) _____

_____

____ 9. Do you have any difficulty breathing. _____

____ 10. Has a doctor ever told you that your cholesterol or triglyceride levels were high? If so, when _____
Do you know what the level was? _____

____ 11. Do you have any physical limitations which prevent you from walking at a brisk pace? _____

____ 12. Has a doctor ever told you to limit your physical activities? _____

____ 13. Do you have any infections? _____

____ 14. List any medications you are currently taking which have been prescribed by a physician _____

_____

____ 15. List any self-administered medications you are currently taking? _____

_____

____ 16. Date of last physical examination. _____
Was it normal? _____ If not, what was abnormal?_____

_____

____ 17. Date of last electrocardiogram _____
Was it normal? _____

____ 18. Date of last cholesterol and triglycerides blood tests

_____

Do you remember the results? _____

Smoking

Have you ever smoked cigarettes, cigars or a pipe? Yes ____ No ____

Do you presently smoke? Yes ____ No ____

If you did or do smoke cigarettes, how many/day?_____
How many years? _____

If you did or to smoke cigars, how many/day? _____
How many years? _____

If you did or do smoke a pipe, how many pipefuls per day _____
How many years? _____

If you have quit smoking, when was it? Month _____
Year _____

Any additional information regarding your health status that you wish to make known?_____

_____

_____

APPENDIX B

MINNESOTA METABOLIC ACTIVITY INDEX

INTERVIEW-QUESIONNAIRE FORM

university of minnesota memo

date November 5, 19 79

to Ms. Susan J. Quaal

from David Jacobs

Enclosed is a copy of the Minnesota Leisure Time Physical Activity Questionnaire as you requested in your letter to me of October 23, 1979

PART IV – LEISURE TIME PHYSICAL ACTIVITIES

Listed below are a series of Leisure Time Activities. Related activities are grouped under general headings. Please read the list and check "yes" in column 3 for those activities which you have performed in the last 12 months, and "no" in column 2 for those you have not. Do not complete any of the other columns.

09443 Due 5-26

To be completed by participant — ACTIVITY (1)	Did you perform this activity? No (2)	Yes (3)	For Clinic Personnel Use Only — Month of Activity (Jan Feb Mar Apr May Jun July Aug Sep Oct Nov Dec)	Average number of times per month (27)	Time per occasion Hrs.	Min. (28)	DO NOT WRITE IN THIS SPACE (25) (34)	
SECTION A: Walking and Miscellaneous								
Walking for Pleasure							010	SKIP 09-SNO
Walking to Work							020	
Using Stairs When Elevator is Available							030	
Cross Country Hiking							040	
Back Packing							050	
Mountain Climbing							060	
Bicycling to Work and/or for Pleasure							115	
Dancing – Ballroom and/or Square							125	
SECTION B: Conditioning Exercise								
Home Exercise							150	
Health Club Exercise							160	
Jogg/Walk Combination							180	
Running							200	
Weight Lifting							210	
SECTION C: Water Activities								
Water Skiing							220	
Sailing in Competition							235	
Canoeing or Rowing for Pleasure							250	
Canoeing or Rowing in Competition							260	
Canoeing on a Camping Trip							270	
Swimming (at least 50 ft.) at a Pool							280	
Swimming at the Beach							295	
Scuba Diving							310	
Snorkeling							320	
SECTION D: Winter Activities								
Snow Skiing, Downhill							340	
Snow Skiing, Cross Country							350	
Ice (or Roller) Skating							360	
Sledding or Tobogganing							370	
SECTION E: Sports								
Bowling							390	
Volley Ball							400	
Table Tennis							410	
Tennis, Singles							420	
Tennis, Doubles							430	
Softball							440	
Badminton							450	
Paddle Ball							460	

09442
Dup
4-25

To be Completed by Participant — ACTIVITY (1)	Did you perform this activity? No (2)	Yes (3)	For Clinic Personnel Use Only — Month of Activity: Jan Feb Mar Apr May Jun July Aug Sept Oct Nov Dec	Average number of times per month	Time per occasion: Hrs.	Min.	DO NOT WRITE IN THIS SPACE	
SECTION E: Sports (Continued)				27	28		24	
Racket Ball							470	SKIP 29 - END
Basketball; Non-Game							480	
Basketball; Game Play							490	
Basketball; Officiating							500	
Touch Football							510	
Handball							520	
Squash							530	
Soccer							540	
GOLF:								
Riding a Power Cart							070	
Walking, Pulling Clubs on Cart							080	
Walking and Carrying Clubs							090	
SECTION F: Lawn and Garden Activities								
Mowing Lawn with Riding Mower							550	
Mowing Lawn Walking Behind Power Mower							560	
Mowing Lawn Pushing Hand Mower							570	
Weeding and Cultivating Garden							580	
Spading, Digging, Filling in Garden							590	
Raking Lawn							600	
Snow Shoveling by Hand							610	
SECTION G: Home Repair Activities								
Carpentry in Workshop							620	
Painting Inside of House, Includes Paper Hanging							630	
Carpentry Outside							640	
Painting outside of House							650	
SECTION H: Fishing and Hunting								
Fishing from River Bank							660	
Fishing in Stream with Wading Boots							670	
Hunting Pheasants or Grouse							680	
Hunting Rabbits, Prairie Chickens, Squirrels, Raccon							690	
Hunting Large Game: Deer, Elk, Bear							710	
SECTION I: Other Activities								

If any question on this form is not clear, ask for clarification at the time of your examination. If you have not answered questions on this form, please inform someone at the clinic at the time of your examination.

APPENDIX C

LETTER OF SUBJECT ACCEPTANCE INTO STUDY

Ms. Susan J. Quaal, RN CVS BSN
Nurse Researcher-Cardiovascular
Screening Project
569 10th Avenue
Salt Lake City, Utah 84103
December 14, 1979

Dear _____

Thank you kindly for consenting to participate in a research study to assess for risk factors for cardiovascular disease amongst the group of office employees at Bell Telephone Co. I reviewed your health history that you completed and found that there is no past medical history which would make you ineligible to participate in the study. The time for which you registered to have the actual physical data collected at the YMCA was: _____.

The YMCA is located at: 737 East 200 South, SLC. Please enter at the EAST entrance, turn right and report to the CHICAGO BRIDGE ROOM (the double-doored room on your right), where we will begin your cardiovascular assessment. Here, again is a recap of the screening procedure you will be subjected to:

Station I: Weight recording, blood pressure and a sample of blood will be drawn from your arm for triglycerides and cholesterol measurement (to be run later in the morning at LDS Hospital lab).

Station II: Skin-fold thickness recording (to be used to compute total body fat).

Station III: The Fisher-Fairbanks Walking Fitness Test will be administered. A resting pulse rate will be recorded, you will walk 100 ft. over 14-15 seconds continuously for 5 minutes and your pulse will then be recorded again (your pre and postwalking pulses will be used in a regression equation formula to calculate oxygen utilization by your body).

By the first week in January, I will send you copies of your lab work and also to your physician, if you've indicated on your health questionnaire. It would be helpful if you would wear a short-sleeved shirt the morning of the testing and it is imperative that you DO NOT EAT, DRINK OR SMOKE ANYTHING FOR 12 HOURS PRIOR TO THE TIME YOUR BLOOD IS DRAWN!!!!!!!!!

If you have any questions prior to the data collection, please feel free to contact me: Susan Quaal, RN CVS BSN Home: 328-0011
Dept. of Physiological Studies
Univ. of Utah College of Nsg. 581-8272

I'll be contacting you by phone again to schedule an appointment to complete the interview portion of the Activity questionnaire you completed. Thank you very much for your participation and I'm looking forward to meeting you again bright and early at the YMCA.

Sincerely,

Susan J. Quaal, RN CVS BSN
Cardiovascular Nurse Specialist

APPENDIX D

PHYSIOLOGICAL DATA COLLECTION FORM

Subject No: _____

Name: _____

Date: _____

Station I

Blood Pressure _____ mm Hg

Station II

Weight _____ lbs

Chest SKINFOLD MEASUREMENTS _____ mm

Axilla _____ mm

Conversions

Chest _____ - Axilla _____ = Density _____ gm/cc

Percent Body Fat _____ %

Station III

Lipid Profile (✓ when drawn) _____

Cholesterol _____ mg %

Triglycerides _____ mg %

LDL _____ mg %

VLDL _____ mg %

HDL _____ mg %

Station IV

FISHER-FAIRBANKS WALKING TEST

Pre-Exercise Resting Pulse _____ bpm

Calibrate (100 ft./14-15 seconds) (✓) _____

Post-five minute exercise pulse _____ bpm

Max VO_2 _____ ml/kg/min

APPENDIX E

STATE OF UTAH DEPARTMENT OF HEALTH HYPERTENSION CONTROL PROGRAM STANDARDS FOR BLOOD PRESSURE MEASUREMENT

Scott M. Matheson
Governor

James O. Mason, M.D., Dr.P.H.
Executive Director
801-533-6111

DIVISIONS
Community Health Services
Environmental Health
Family Health Services
Health Care Financing and Standards

OFFICES
Administrative Services
Health Planning and Policy Development
Medical Examiner
State Health Laboratory

STATE OF UTAH
DEPARTMENT OF HEALTH
DIVISION OF COMMUNITY HEALTH SERVICES
150 West North Temple, P.O. Box 2500, Salt Lake City, Utah 84110

BUREAU OF CHRONIC DISEASE CONTROL
Gayle E. Reiber, M.P.H., Director
Room 460 801-533-6141

January 4, 1980

Sue Quaal, RN, CVS, BSN
569 10th Avenue
Salt Lake City, UT 84103

Dear Ms. Quaal:

This letter will certify the participation of the Utah State Department of Health, Hypertension Control Program staff in your thesis research project. The Hypertension Control staff, consisting of Joan Ware, R.N., B.S.N., Nadine Fishbeck, R.N., B.S.N., and Staci Morgan, B.S. Health Education, has been standardized in blood pressure measurement technique, according to the attached protocol. The staff has participated in the following research projects:

Project	Principal Investigator
Genetic Predisposition to Hypertension	Roger R. Williams, M.D. University of Utah College of Medicine
Familial Predisposition to Coronary Artery Disease	Roger R. Williams, M.D. University of Utah College of Medicine Frank Yanowitz, M.D. LDS Hospital Cardiology
Effect of Weight Loss on Blood Pressure	Susan Mendenhall, R.D. Utah Heart Association
Effect of Combination of Weight Loss and Exercise on Blood Pressure	Robin Beck, R.N., B.S.N. University of Utah College of Nursing Utah Heart Association
Variables in the Presence or Absence of "Hot Flashes" in Menapausal and Post-Menapausal Females	Carla Biehl, R.N., B.S.N. University of Utah College of Nursing
Performance Testing of an Automated Indirect Blood Pressure Measurement System	John A. Burkart, Ph.D. UBTL, University of Utah Research Institute

In all cases the staff has been consistant, without significant variance.

If further information is desired, please call me at 533-6141.

Sincerely,

Joan Ware

Joan Ware, R.N., Director
Hypertension Control Program
Bureau of Chronic Disease Control

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Enclosure

UTAH STATE DIVISION OF HEALTH
DISEASE CONTROL BRANCH
BUREAU OF CHRONIC DISEASE CONTROL

HYPERTENSION CONTROL PROGRAM

STANDARDS FOR BLOOD PRESSURE MEASUREMENT OF ADULTS SECOND REVISION, 1979.

I. GENERAL CONSIDERATIONS:

Cardiovascular disease is the primary cause of death in Utah and the United States, accounting for nearly one million deaths, or 53 percent of all deaths in the United States (1), and 42.4 percent in Utah (2). A major risk factor and contributor to this mortality is high blood pressure, which leads to heart disease, kidney disease and stroke (3-5).

The American Heart Association estimates that approximately 33,740,000 people have hypertension, or one in six adults (6). According to baseline data from National Heart, Lung, and Blood Institute (NHLBI), at most, only 40 percent of the hypertensive population is adequately treated or controlled (7). Using national data, this means that there are approximately 132,302 adults in Utah with treated or untreated hypertension.

The 1971-1974 National Health Survey shows that proportionately more women than men currently have hypertension. High blood pressure is more prevalent in

rural areas and incidence declines as levels of education and family incomes increase. Hypertension affects more blacks than whites and is somewhat more prevalent in the Southern United States. Little reliable data is available regarding hypertension prevalence among Spanish, Native or Asian American populations (9).

It has been demonstrated by life insurance companies, that years of life are lost and unnecessary morbidity results from uncontrolled high blood pressure (10). This has prompted public health emphasis on screening, early detection, maintenance and education programs for high blood pressure control (11).

With such widespread interest in and reliance on the procedure of blood pressure measurement, standardization of all personnel and equipment is necessary to minimize the major sources of error which contribute to variation in blood pressure measurement.

This standardization should include the following:

A. Selection of quality blood pressure measuring equipment and proper maintenance of equipment, including accuracy checks.

B. Instruction and accreditation in the measurement techniques recommended by the American Heart Association and The National High Blood Pressure Coordinating Committee (12, 13) of all personnel directly involved in blood pressure measurement.

C. Organization of clinical areas to allow adequate space for the following:

- Registration
- Five minute rest period
- Health education
- Blood pressure determination
- Waiting time and repeat blood pressure measurement as indicated
- Counseling

II. PHYSIOLOGY OF BLOOD PRESSURE:

A. Background

When a small artery is cut, blood will spurt from the point of injury to a considerable height. The column of blood will rise and fall, reaching its maximum height during cardiac systole (contraction) and its minimum during diastole (relaxation). This indicates that the blood within the arteries is under pressure and that the pressure varies with the phase of the cardiac cycle. The peak, which occurs during ventricular systole, is called systolic pressure. The minimum value, which occurs during ventricular diastole, is known as diastolic pressure. The arithmetic difference between systolic and diastolic pressure is called pulse pressure.

Blood pressure is measured in terms of the height of the column of mercury that it is capable of supporting. The values for systolic and diastolic pressures are customarily given as systolic pressure over diastolic pressure, or 120/80.

Blood pressure is the result of the pumping action of the heart, which empties blood into a closed system of elastic vessels. The volume capacity of this system does not remain constant. It is changed through the elastic stretch of the vessels and by variations in the caliber of the vessels in response to nervous and chemical stimuli and fluid volume. Since, during ventricular systole (contraction), blood is forced into the highly elastic arterial system faster than it can escape into the capillaries and veins, the arteries are stretched to greater capacity. The elastic recoil of the arterial walls forces the blood forward through the vessels at a constantly decreasing pressure until the arteries regain the presystolic caliber. As blood is forced out of the heart, it travels through the aorta to arteries, which progressively become smaller arterioles. The blood then passes through capillaries, to the venules; to the larger veins and is returned to the heart (14).

B. The Sounds of Korotkoff

Distinct blood pressure sounds were first described by a Russian physician named Korotkoff. He identified the following five phases in blood pressure:

Phase	Description
1. First Phase	A clear tapping sound; the onset of the sound for two consecutive beats is considered systolic blood pressure (15).
2. Second Phase	The tapping sound, followed by a murmur. Usually 10-15 mm.Hg. below first phase and lasting for 14-20 mm.Hg. In some instances, such as when the cuff is inflated too slowly, part or all of the sounds of this phase may be absent, resulting in a period of silence known as an auscultatory gap (16).
3. Third Phase	A loud, crisp tapping sound.
4. Fourth Phase	Abrupt, distinct muffling of sound, gradually decreasing in intensity.
5. Fifth Phase	The disappearance of sound, considered diastolic blood pressure (17).

In some individuals, the point at which sound disappears may be very low, or near 0 mm.Hg. This occurs when high velosity blood flow exists, as in exercise, anemia, fever, thyrotoxicosis or arteriovenous fistulas (15), or in clients where cardiac output is high, as with children and pregnant women (13). In these cases, record the onset of sound/muffle/disappearance of sound, i.e., 120/80/0 mm.Hg. (12).

III. EQUIPMENT:

Maintenance of equipment, familiarity and availability of adequate cuff

sizes are the primary considerations in controlling errors due to the nature of the equipment. Equipment used in community blood pressure screening clinics include:

1. Sphygmomanometer
   a. Standard Mercury Baumanometer
   b. Aneroid Sphygmomanometer

A mercury sphygmomanometer is preferred, but a properly calibrated aneroid manometer may be used provided it is regulary recalibrated (12). Good equipment maintenance features calibration and standardization every six months for any measuring device. In interpreting standardization results, it should be noted that ± 3 mmHg at any pressure level is the limit of tolerance set for sphygmomanometers by the National Bureau of Standards (19).

2. Cuff Sizes
   a. Standard adult
   b. Large adult
   c. Child
   d. Infant
   e. Thigh (recommended)
3. Dual Head Stethoscope

IV. MEASUREMENT TECHNIQUES

The technique for blood pressure determination recommended by both the National High Blood Pressure Coordinating Committee and the American Heart Association is known as the pulse obliteration method, maximum inflation level or palpatory-auscultatory method (12, 13). Employment of the following techniques will contribute to the overall skill, precision, and accuracy with which health care providers measure blood pressure.

GUIDELINES FOR BLOOD PRESSURE MEASUREMENT

	TECHNIQUE	RATIONALE
1.	Individuals desiring a blood pressure measurement need to complete the data collection and consent form (11).	
2.	Clients should be seated for five minutes and requested to delay smoking or drinking beverages containing caffeine, as this may alter blood pressure readings (11, 12)	Vasoconstrictive substances may increase the blood pressure (11, 15).
	Screener should consider other factors that may increase blood pressure, i.e. climate, antihistamine or other medications, bladder distention, recent exposure to high altitude and current or recent stress (12).	Cold climates may cause vasoconstriction and increase blood pressure. Increased physiological demand as with high altitudes may cause increased blood pressure (12). Physiologic and emotional stress stimulate the orienting reflex or defense reaction and elevate blood pressure by the following mechanism: vasodilatation in muscle vasoconstriction in skin and intestines increase in heart rate increase in cerebral blood flow increase in cardiac output increase in blood pressure Bladder distention produces a fairly generalized vasoconstriction reflexly through the spiral cord (19).
3.	Client should be sitting up straight with both feet flat on the floor as the procedure is explained to him.	Crossing legs can increase blood pressure. Alteration of body position alters blood pressure. Slouching will elevate both levels (20).
4.	Expose the upper right arm. Use the left arm only if there is right arm injuries, trauma or severe dermatitis.	Baumanometers are designed for right arm measurements (21). Since brachial artery pressure may normally differ by as much as 10 mm.Hg. and is usually higher in the right arm (22), use of right arm for standardization and consistency of readings is recommended.
5.	Make certain upper right arm is at the level of the heart, elbow slightly flexed forearm, with the palm facing upwards and firmly supported on a flat surface.	If upper arm is lower than heart level, blood pressure could increase by as much as 10 mmHg in both systolic and diastolic readings (12, 23).

6. The blood pressure cuff should be applied so the cuff is one inch above the natural crease (anticubital fossa) with the inflatable bladder centered over the brachial artery.	This space allows proper placement of stethoscope. If a portion of the head of stethoscope is placed under the cuff, it may cause uneven cuff pressure and distort the reading (24). It also reduces the possibility of bumping the tubing or the cuff.
7. Wrap the cuff snugly around the upper arm area.	The bladder of a loosely wrapped cuff will balloon and decrease the effective width causing an elevated reading (20).
8. Be certain you are using the proper size cuff. The width of the cuff for an adult should be 20 percent greater than the diameter of the arm and for children the cuff width should be two-thirds the length of the upper arm (12). For obese adults, use a special large arm cuff. The bladder should encircle half the limb (25, 26). Many cuffs now have straight line markings on adjoining surface of the cuff and when the markings overlap, or fall within the prescribed area, this indicates a properly sized cuff.	
9. Attach cuff to the standard Baumanometer.	
10. Inflate cuff while palpating the radial artery pulse until the pulse is obliterated (12). Make note of the pulse obliteration level and deflate. This level will closely approximate the systolic blood pressure.	This method eliminates the problem of an ascultatory gap, the temporary disappearance of second phase blood pressure sounds. Phase one Koratkoff sounds are heard over the brachial artery when cuff pressure is high and disappear as pressure is reduced, (phase two) reappearing at a lower level (phase three). If the screener does not know how high to properly inflate the cuff, it is possible the first sounds heard could be the reappearance of sounds in the third phase, not the first phase. This would result in underestimation of systolic blood pressure by as much as 40 mm.Hg. (12, 27). The pulse obliteration technique also alerts the screener to irregularities of heart rhythm, which would affect interpretation of blood pressure readings (11). Over inflation of the cuff may cause a spasm of the vessels, resulting in increased peripheral resistance, which creates increased myocardial taxation, resulting in increased blood pressure (24). Unnecessary elevations of cuff pressure may also cause pain to client, resulting in a stress response and an elevated reading (19, 24).

Calculate peak inflation by adding 30 mm.Hg. to the reading at which the radial pulse disappeared (12). Example: Pulse Obliteration point 124 mmHg + 30 Peak Inflation Level 154 mmHg	
11. Palpate the brachial artery pulse. Place the stethoscope (diaphram for adults and bell for children) over the brachial artery. Avoid allowing the stethoscope to bump the cuff or tubing. Make sure the entire surface of the stethoscope is against the surface of the arm. Apply as little pressure on the head of the stethoscope as possible.	Careful placement of the stethoscope permits the greatest audibility of brachial arterial sounds (12). Heavy pressure distorts the artery and produces sounds heard below diastolic pressure (12).
12. Allow 60 seconds to elapse between pulse obliteration and auditory measurement.	This will allow release of blood trapped in the veins (12).
13. Rapidly inflate the cuff to peak inflation level.	An auscultatory gap is more likely to occur if the blood pressure cuff is inflated too slowly (28).
14. Deflate the cuff at 2 mm.Hg./sec.	If deflation is slower than 2 mm.Hg./sec. venous congestion develops and diastolic reading could be elevated (20). If deflation is more rapid than 2 mm.Hg./sec. the observer may err in identifying faint sounds either at onset or diappearance of sounds.
Avoid re-inflating the cuff after deflation has begun.	Venous return will engorge the forearm with blood and produce a loss of clarity of diastolic endpoint (28).
Listen for the onset and disappearance of Korotkoff sounds. Do not be confused by bounces in the column of mercury or of the needle on the aneroid dial - note auditory sounds not visual cues. Continue the deflation at this rate 10 mm.Hg. past the disappearance of sound, then deflate rapidly.	If the cardiac rhythm is irregular, rapid deflation of the cuff upon initial disappearence of sound may result in elevated diastolic reading (29).
15. Record the onset of sound and the disappearance of sound on the appropriate form (11).	

In recording remember:

-All readings are rounded up to the nearest even digit.

-All readings are read at the top of the meniscus.

-Systolic reading is the point at which the initial tapping sound is heard for at least two consecutive beats (12).

-Diastolic reading is 2 mm.Hg. below the last sound heard, which is the disappearance of sound (11).

-If uncertain, wait at least one minute and repeat (13).

If the sounds were faint, the following techniques may help to enchance sounds:

-Inflate the cuff more rapidly.

-Recheck the placement of stethoscope over the brachial artery.

-Eliminate extraneous noise.

-Ask client to elevate his/her arm for 10-15 seconds, then rapidly inflate the cuff to peak inflation level while arm is elevated, lower arm and proceed with measurement (16).

Should you find it necessary to take a thigh blood pressure, have the client lie on his/her abdomen, palpate the popliteal pulse and apply the center of the bladder 1-1½ inches above the natural crease in the knee. While palpating the popliteal pulse, inflate the cuff until the pulse is obliterated. Deflate, wait 60 seconds, then inflate to 30 mm.Hg. higher than the pulse obliteration point. Using the techniques described previously, deflate at 2 mm.Hg/sec. or 2 mm.Hg. per heartbeat, noting onset and disappearance of sound. The systolic reading for a thigh blood pressure averages 10-40 mm.Hg. higher than that in the arm, but the diastolic readings are essentially the same (12).

Remember to record the individual's blood pressure on <u>both</u> the data collection form and the wallet card.

V. BLOOD PRESSURE GUIDELINES AND STANDARDS FOR REFERRAL:

If the individual's blood pressure was elevated for his/her age, ask him/her to sit quietly for 5-10 minutes and <u>repeat</u> the measurement (20). Several factors could result in an initially elevated blood pressure, including

arterial spasms upon initial compression of the cuff, anxiety, and apprehension (20).

If the second blood pressure reading is elevated, follow-up is indicated. Refer to the guidelines below when scheduling a repeat appointment. Be careful to describe the elevated blood pressure reading as "elevated". Avoid using the terms "high blood pressure" and "hypertension", since these refer to diagnostic, not screening procedures.

BLOOD PRESSURE GUIDELINES: STANDARDS FOR REFERRAL (11, 15, 20)
UTAH STATE DIVISION OF HEALTH
BUREAU OF CHRONIC DISEASE CONTROL

First Screening

Class I	Everyone less than	140/90 mm.Hg.	Recheck every year
Class II	Under 50 years of age	140/90 or greater	Recheck in 1-2 weeks
Class III	Age 50 and over	166/90 or greater	Recheck in 1-2 weeks
Class IV	Anyone over	200/120 or greater	Refer to physician immediately

To prevent over-referral, a client with an elevated blood pressure, Class II or III, should have two blood pressure measurements on three different occasions, using the standards above before initiating physican referral. This will eliminate false positives and provide the physician with a series of measurements over a period of time, which can help in treatment decisions. There will be special situations which will require the observer to make judgments beyond those outlined in these guidelines. The judgments are both advised and encouraged. (See Figure 1.)

VI. FOLLOW-UP:

Follow-up and referral are ethical responsibilities of any hypertension detection program. All individuals needing follow-up and/or referral should be followed until they are under proper blood pressure control. Several points to keep in mind for follow-up of elevated blood pressures include:

-Be certain to obtain the individual's name, address, phone number, blood pressure measurement and signed consent.

-Assign a follow-up appointment for the individual.

-Provide a brochure or other printed materials, giving information on high blood pressure to individuals with elevated blood pressure.

-Make certain the individual receives a wallet card with blood pressure and form number recorded.

-Remind the individual of the appointment, either by phone call or letter within 24 hours of the scheduled appointment.

-Should the individual not keep the follow-up appointment, a telephone inquiry and rescheduling of the appointment is desirable.

-Send letters to those not keeping follow-up appointments and include additional educational information and a follow-up clinic schedule. Request clients to call you if they have obtained care for their elevated blood pressure.

If these suggestions and quidelines are followed, accuracy can be insured and a useful, lifesaving service provided to the community.

REFERENCES

1. Levy, Robert I. Progress in Prevention of Cardiovascular Disease. Preventive Medicine, 1978.

2. Annual Report of Utah Vital Statistics, Utah State Division of Health, Salt Lake City, 1978.

3. Characteristics of Persons with Hypertension, U.S. Department of Health, Education and Welfare. Publication No. 79:1549, 1978.

4. Kadnel, W.B., Dawber, T.R., et al. Vascular Disease of the Brain - Epidemiologic Aspects: The Framingham Study. A.J.P.H., 1965.

5. Stamler, Jeremiah, and Stamler, Rose, and Pullman, Theodore N. (Ed.) Build and Blood Pressure Study, 1959. The Epidemiology of Hypertension. New York and London: Grune and Stratton, 1967.

6. Heart Facts 1979, American Heart Association, 1978.

7. Program Outlines for State Hypertension Control Program, Department of Health Education and Welfare, 1978.

8. Brockert, John E. 1978 Population Estimates for Utah. Utah Economic and Business Review. 1978.

9. Blood Pressure Levels of Persons 6-74 Years, United States, 1971-1974, U.S. Department of Health, Education and Welfare, Publication No. 78:1648, September, 1977.

10. Society of Actuaries, Build and Blood Pressure Study, Chicago, 1959.

11. Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure, DHEW Publication No. (NIH) 78-1088.

12. Recommendations for Human Blood Pressure Determination by Sphygmomanometers, American Heart Association, 1967.

13. A Review of Techniques for Training Program for Measurement of Blood Pressure. High Blood Pressure Information Center, 1978.

14. Guyton, Arsdur. Textbook of Medical Physiology, Philadelphia: W.B. Saunders, 1971.

15. Hurst, J. Willis. The Heart, New York: McGraw Hill, 1978.

16. Ravin, Abe. The Clinical Significance of the Sounds of Korotkoff, an audio cassette program, University of Colorado School of Medicine.

17. Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure, High Blood Pressure Coordinating Committee, National High Blood Pressure Education Program, 1976.

18. Wyman, W. F. The Aneroid Sphymomanometer - An Assessment of Accuracy. Cardiovascular Medicine, July, 1978.

19. Pickering, George. High Blood Pressure, 2nd Edition, New York: Grune and Stratton, Inc., 1968.

20. Burch, G. E. and DePasquale, N. P. Primer of Clinical Measurement of Blood Pressure. St. Louis: The C.V. Mosby Co., 1962.

21. W. A. Baum Co., Inc., 1969.

22. Bates, Barbara. A Guide to Physical Examination. Philadelphia: J. B. Lippincott Company, 1974.

23. Mitchell, P. L., Parlin, R. W., and Blackburn, H. Effects of Vertical Displacement of the Arm on Indirect Blood Pressure Measurement. New Eng J of Medicine, 1964.

24. Lancour, Jane. How to Avoid Pitfalls in Measuring Blood Pressure. AJN, 1976.

25. Geddes, M. E. and Wjost, S. J. The Error in Indirect Blood Pressure Measurement with the Incorrect Size of Cuff. American Heart J. 1978.

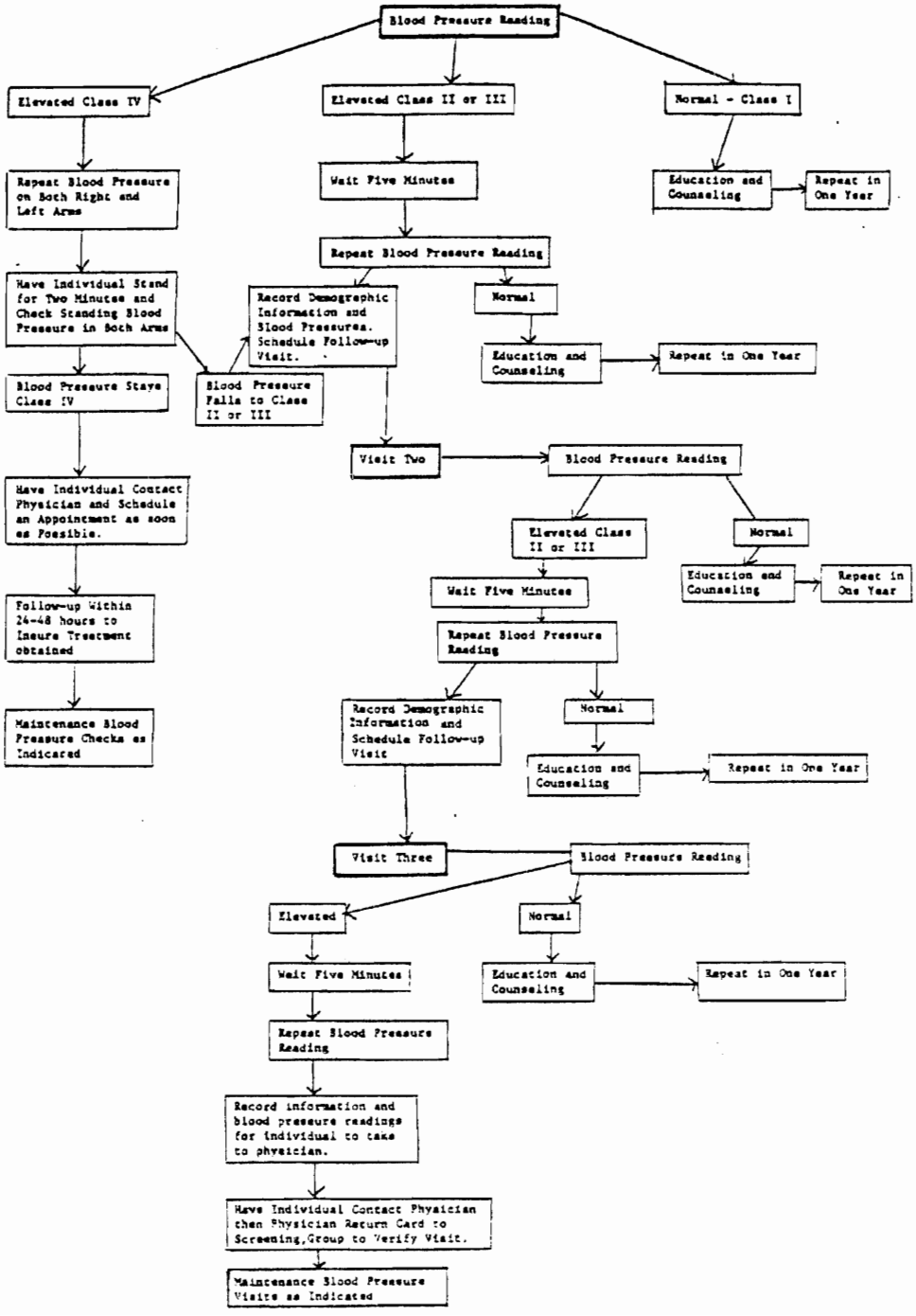
26. If the Cuff Doesn't Fit, Adjust the Reading. Cardiovascular Medicine, November, 1978.

27. Askey, John Martin. The Auscultatory Gap in Sphygmomanometry. Annuals of Internal Med, 1974.

28. Blood Pressure: Its Control and Measurement. American Heart Association, 1977.

29. Skidmore, Elaine and Marchall A. Towards a More Accurate Measurement of Blood Pressure. Nursing Times, 1976.

FIGURE 1.


Blood Pressure Reading
Elevated Class IV
Elevated Class II or III
Normal - Class I
Repeat Blood Pressure on Both Right and Left Arms
Wait Five Minutes
Education and Counseling
Repeat in One Year
Repeat Blood Pressure Reading
Have Individual Stand for Two Minutes and Check Standing Blood Pressure in Both Arms
Record Demographic Information and Blood Pressures. Schedule Follow-up Visit.
Normal
Education and Counseling
Repeat in One Year
Blood Pressure Stays Class IV
Blood Pressure Falls to Class II or III
Visit Two
Blood Pressure Reading
Have Individual Contact Physician and Schedule an Appointment as soon as Possible.
Elevated Class II or III
Normal
Education and Counseling
Repeat in One Year
Wait Five Minutes
Follow-up Within 24-48 hours to Insure Treatment obtained
Repeat Blood Pressure Reading
Record Demographic Information and Schedule Follow-up Visit
Normal
Education and Counseling
Repeat in One Year
Maintenance Blood Pressure Checks as Indicated
Visit Three
Blood Pressure Reading
Elevated
Normal
Wait Five Minutes
Education and Counseling
Repeat in One Year
Repeat Blood Pressure Reading
Record information and blood pressure readings for individual to take to physician.
Have Individual Contact Physician then Physician Return Card to Screening Group to Verify Visit.
Maintenance Blood Pressure Visits as Indicated

APPENDIX F

LDS HOSPITAL PATHOLOGY LABORATORY PROCEDURE MANUAL FOR ANALYSIS OF BLOOD PLASMA LIPIDS

LDS HOSPITAL
A MAJOR REFERRAL CENTER
325 Eighth Ave., Salt Lake City, Utah 84143 [illegible]

Department of Pathology
Fred Mival, M.D., Ph.D., Chairman

February 15, 1980

Susan Quaal
569 10th Avenue
Salt Lake City, Utah 84103

Dear Susan:

This letter will give you my permission to use the LDS Hospital Pathology Laboratory Procedure Manual for Cholesterol Fractionation in your study. This is the procedure which is used in our laboratory for the determination of Cholesterol, Triglyceride, HDL, LDL, VLDL, as part of the Lipid Profile which we perform. This is the procedure which was used on the specimens that you submitted as part of your study.

You asked about the invalidation of results if the Triglyceride is over 400. Since VLDL is a calculated value based on the results of the Triglyceride determination, results over 400 give a elevated VLDL which does not represent true VLDL levels. This has been shown in several studies one of which was referenced in the Procedure Manual procedure.

It was a pleasure to be of some help in your study and if we can ever be of help to you in the future please feel free to let me know.

Respectfully,

Acel Kent Thacker

Acel Kent Thacker
Chemistry Supervisor

PROCEDURE: FRACTIONATED CHOLESTEROL

PRINCIPLE: The addition of ISOPOL precipitating reagent lowers serum pH to the isoelectric point of low density lipoprotein (ph = 5.7). Phosphotungstate forms an insoluable complex with LDL which can be removed by centrifugation. HDL values can then be determined by a cholesterol method. Triglyceride and cholesterol methods are contained in the DuPont ACA Instruction Manual. It has been observed that an inverse relationship exists between the levels of serum HDL and coronary heart disease, while a direct relationship occurs between LDL and CHD risk.

PATIENT PREPARTION: Patient should fast for 12 hours before specimen is drawn.

SPECIMEN: Serum, preferably unhemolyzed, is used for fractionated cholesterol determination. Sample may be stored up to three days at 2-30 C.

TRANSPORTATION: Usual method is sufficient.

REAGENTS: A. ISOPOL Precipitating Reagent
1. 0.4% phosphotungstic acid in a solution containing buffers and stabilizers.
2. Precautions: Causes irritation. Avoid contact with eyes, skin, clothing. Do not ingest.
3. Stable until expiration date. Store at 2-30 C.
4. Deterioration: The reagent should be clear. If a cloudiness develops, the reagent may have deteriorated and should not be used.

B. DuPont ACA Cholesterol pack. (see ACA manual)
C. DuPont ACA Triglyceride pack. (see ACA manual)

CONTROLS: LDS Serum Pool

STANDARDS: ACA triglyceride and cholesterol standard

DIRECTIONS FOR STANDARDIZATION: Outlined in ACA Instrument Instruction manual.

PROCEDURE:
1. Perform cholesterol and triglycerides on downstairs ACA using DuPont ACA triglyceride and cholesterol packs.
2. Pipette 0.5 ml serum into small labeled test tube.
3. Add 0.5 ml ISOPOL precipitating reagent and mix well.
4. Centrifuge for 10 minutes at 750 X g.
5. Separate supernatant from precipitateand measure HDL by running precipitate on ACA with a cholesterol pack.
6. Observe whether serum is clear, cloudy or milky.
7. HDL appears stable for 48 hours at room temperature.
8. Precipitated serum need not be centrifuged immediately, but should be done within two hours of adding ISOPOL.

.LABORATORY PROCEDURE MANUAL (cont...) FRACTIONATED CHOLESTEROL

CALCULATIONS: Cholesterol: Read directly from ACA printout.
LDL: Cholesterol - (VLDL + HDL)
HDL: Printout value of cholesterol determination performed on supernatant X 2.
VLDL: Triglyceride value divided by 5
Triglyceride: Read directly from ACA printout.

DERIVATION OF RESULTS: See calculations.

SOURCES OF ERROR: Samples precipitated when cold may show values slightly lower than those done at room temperature.

CRITERIA OF UNACCEPTABLE RESULTS: Triglyceride values over 400 invalidate low density lipoprotein and very low density lipoprotein, so do not report these values when triglyceride values exceed 400. High density lipoprotein values are reported, along with cholesterol and triglyceride values. A coded comment "FR" will tell the physician why the test was reported out as it was. Pool values out of range invalidate the test, also.

NORMALS:

	Male	Female
Cholesterol	150 - 275 mg/100 ml	150 - 275 mg/100 ml
LDL	62 - 178 mg/100 ml	66 - 185 mg/100 ml
HDL	29 - 61 mg/100 ml	38 - 75 mg/100 ml
VLDL	0 - 40 mg/100 ml	0 - 40 mg/100 ml
Triglyceride	50-- 200 mg/100 ml	50 - 200 mg/100ml

REFERENCES: "ISOPOL Precipitating Reagent" (product enclosure), Data Medical Associates.
Castelli, W.P., et al, Circulation 55: 767 (1967).
Gordon, J., et al, AMJ Med, 62: 702 (1977).

PREPARED BY DR DATE 3/15/79 REVIEWED BY [illegible]

When you get a triglyceride of 400 or more, run the HDL and report the results, but do not calculate the VLDL & LDL as these values will not be correct. Thanks

Chem. Supervisor

DMA

ISOPOL™ Precipitating Reagent

(For the Quantitative Separation of High-Density Lipoproteins from Serum)

SUMMARY AND EXPLANATION:

Recently, Castelli and co-workers (1) have established an inverse relationship between serum high-density lipoprotein (HDL) cholesterol and the risk of coronary heart disease. Total and HDL cholesterols, in conjunction with a triglyceride determination, provide valuable information for the prediction of coronary heart disease (2).

The DMA ISOPOL Precipitating Reagent offers a means for the rapid and complete separation of HDL from other serum lipoproteins. The isolated HDL fraction can be then analyzed for cholesterol content. The separation is based on isoelectric-polyanionic precipitation of low-density lipoproteins (LDL). The precipitating reagent does not contain metal ions, and differs from first generation precipitating reagents that use divalent metal ions (e.g. Mn^{++}, Mg^{++} Ca^{++}) to form complexes with LDL. The molecular interactions involving metal ions are relatively weak and depend on time, temperature, ionic strength and metal-binding agents. The polyanion component of ISOPOL Precipitating Reagent acts directly on LDL to form strongly associated, insoluble complexes. This strong interaction assures rapid and complete separation of HDL from other serum lipoproteins at a reduced polyanion concentration. The precipitation is immediate at ambient temperature.

PRINCIPLE:

ISOPOL Precipitating Reagent uses the well established precipitating properties of phosphotungstate (3). Upon the addition of ISOPOL Precipitating Reagent, serum pH is lowered to the isoelectric point of low-density lipoprotein (pH = 5.7), where the molecules have over-all electrical neutrality. At this pH phosphotungstate forms an insoluble complex with LDL which can be removed by centrifugation.

REAGENT. FOR IN VITRO DIAGNOSTIC USE

ISOPOL Precipitating Reagent. REACTIVE INGREDIENT: 0.4% phosphotungstic acid. Buffers and stabilizers added.

PRECAUTIONS

Consumption: Avoid contact with eyes, skin and clothing. Do not ingest.

STORAGE AND STABILITY

Store until expiration date. Store at 2°–30°C.

DETERIORATION

The reagent should be clear. If a cloudiness develops, the reagent is now deteriorated and should not be used.

SPECIMEN COLLECTION:

1. DMA recommends unhemolyzed serum. The specimen need not be fasting.
2. No special additives or preservatives are required.
3. Heparinized plasma should not be used.

SAMPLE STORAGE

The sample may be stored at 2°–30°C. Serum HDL cholesterol is stable at least three days. Samples precipitated while cold may show values slightly lower than samples precipitated at room temperature.

PROCEDURE:

MATERIALS PROVIDED

ISOPOL Precipitating Reagent

MATERIALS REQUIRED BUT NOT PROVIDED

1. Pipettes for accurately dispensing 0.50 mL volume
2. Clinical centrifuge capable of 750 x g.

REACTION CONDITIONS

Precipitation Temperature	Ambient
Precipitation Time	Immediate
Centrifugation Time	10 Minutes at 750 x g
Sample Volume	0.50 mL
Reagent Volume	0.50 mL
Total Volume	1.00 mL

PERFORMANCE OF TEST:

Read entire PERFORMANCE OF TEST section before proceeding with precipitation.

Precipitation

1. Dispense 0.50 mL serum into an appropriately labeled 13x100 mm tube.
2. Add 0.50 mL ISOPOL Precipitating Reagent. Mix well.
3. Centrifuge for 10 mintues at 750 x g (¾ speed for clinical centrifuge).
4. Separate supernatant from precipitate. The supernatant (top) layer contains HDL.

Procedural Note: The precipitated serum does not have to be centrifuged immediately. However, the sample should be centrifuged within two hours after precipitation. The supernatant may develop some turbidity upon standing which does not interfere with the cholesterol assay. (If a flaky precipitate forms, centrifuge the supernatant before sampling.)

STABILITY OF FINAL REACTION PRODUCT

HDL cholesterol in the supernatant appears stable at least 48 hours at controlled room temperature (15°–30°C).

COMMENT ON CHOLESTEROL ASSAY:

DMA designed ISOPOL Precipitating Reagent for use with enzymatic cholesterol reagents. Optimum results are obtained when the supernatant volume used in the assay is 10 X the volume employed for total cholesterol assay. At this level sensitivity approaches 0.1 mg/dL HDL cholesterol if a suitable spectrophotometer is employed (resolution of A = 0.001).

Liebermann-Burchard reagents can be used to assay HDL cholesterol. However, sample volume should not be altered with these procedures and sensitivity is limited.

QUALITY CONTROL:

A pooled serum which has been aliquoted and frozen may be used to quality control the precipitation step. Do not repeatedly freeze and thaw. Control sera with normal and abnormal levels should be employed routinely with the cholesterol assay. DMA suggests the use of Data-Trol A and Data-Trol.

CALCULATION OF RESULTS:

Use the equation below to calculate HDL cholesterol.

$$\frac{\text{Abs. of Unk.}}{\text{Abs. of Std.}} \times \text{Std (mg/dL)} \times 2 \times \frac{\text{TV/SV (HDL)}}{\text{TV/SV (Std.)}} = \text{HDL Cholesterol (mg/dL)}$$

The total volume / sample volume (TV/SV) ratio adjusts for volume difference between standard and supernatant used in the cholesterol assay. If these volumes are the same, this ratio is not required for the calculation. The factor 2 corrects for sample dilution during the precipitation step.

EXAMPLE

Standard (300 mg/dL): Reagent volume = 2.00 mL; sample volume = 0.020 mL; Abs. = 0.590.

Supernatant: Reagent = 2.00 mL; sample volume = 0.20 mL; Abs. = 0.450.

$$\frac{0.450}{0.590} \times 300 \text{ mg/dL} \times 2 \times \frac{2.2/0.2}{2.02/0.02} = 49.8 \text{ mg/dL}$$

For these conditions, the equation reduces to:

$$\frac{0.450}{0.590} \times 65.3 \text{ (constant factor)} = 49.8 \text{ mg/dL}$$

If a direct read-out instrument is employed, set the standard value at 65.3 in the concentration mode.

LIMITATIONS:

HDL cholesterol analysis is intended for the assessment of coronary heart disease risk in apparently normal individuals. Results obtained for patients with hyperlipoproteinemia, liver disease or recent myocardial infarction have no correlation.

EXPECTED VALUES:	Males:	26–63
	Females:	33–75

The above values were established from a study of 28 males and 29 females judged clinically normal by multiphasic testing. DMA recommends that each laboratory establish its own expected range.

INTERPRETATION OF RESULTS:

Serum HDL cholesterol is the best single indicator of coronary heart disease risk in an apparently normal individual (2). Generally, HDL cholesterol levels below 35 mg/dL represent high risk, from 35–55 mg/dL an intermediate risk, and above 55 mg/dL a low risk.

A more accurate assessment can be established by considering the serum total cholesterol to HDL cholesterol ratio (4). These risk factors are sex dependent.

RISK	TOTAL / HDL RATIO	
	MEN	WOMEN
½ Average	3.43	3.27
Average	4.97	4.44
2X Average	9.55	7.05
3X Average	23.99	11.04

BIBLIOGRAPHY:

1. Castelli, W.P., Doyle, J.T., Gordon, T., Hames, C.G., Hjortland, M.C., Hulley, S.B., Kagan, A., and Zukel, W.J., Circulation 55, 767 (1977).
2. Gordon, T., Castelli, W.P., Hjortland, M.C., Kannel, W.B., and Dawber, T.R., Am. J. Med. 62, 707 (1977).
3. Burstein, M., Scholnick, H.R., and Morfin, R., J. Lip. Res. 11, 583 (1970).
4. Castelli, W.P., and Levitas, I.M., Current Prescribing 6, 30 (1977).

aca DU PONT AUTOMATIC CLINICAL ANALYZER

aca
TEST METHODOLOGY

CHOL
CHOLESTEROL

INTENDED USE:

The CHOL pack is used in the Du Pont Automatic Clinical Analyzer (aca) to quantitatively measure total cholesterol in serum.

SUMMARY:

The aca enzymatic cholesterol (CHOL) method is based on the principle first described by Stadtman[1,2] and later adapted by other workers.[3,4,5,6] The aca modifications include the unique use of the chromogen N, N-diethylaniline·HCl to increase the sensitivity of the method.

Split sample comparison between the aca CHOL method and an automated Liebermann-Burchard procedure[7] gave a correlation coefficient of 0.992 (correlation slope = 0.976; correlation intercept = −4.1 mg/dl). Compared to a similar enzymatic procedure, the aca gave a correlation coefficient of 0.976 (correlation slope = 0.960; correlation intercept = 11.4 mg/dl). Typical within-run and day-to-day coefficients of variation were 1.2% and 1.5%, respectively.[4]

The maximum random, systematic and total analytical errors as defined by Westgard, et al.[8] were 7.15, 4.35, and 10.32 mg/dl, respectively, at a cholesterol concentration of 250 mg/dl. The total analytical error was significantly lower than the "accepted performance standard value" of 20 mg/dl.[9]

The enzymatic method selected for the aca offers the advantage of greater specificity than chemical methods and is not subject to interference from bilirubin, hemolysis, lipemia, or reducing substances at levels commonly found in serum. Sample extraction is unnecessary.

PRINCIPLES OF PROCEDURE:

Cholesterol esterase (CE) catalyzes the hydrolysis of cholesterol esters to produce free cholesterol which, along with preexisting free cholesterol, is oxidized in a reaction catalyzed by cholesterol oxidase (CO) to form cholest-4-ene-3-one and hydrogen peroxide. In the presence of horseradish peroxidase (HPO), the hydrogen peroxide thus formed is used to oxidize N, N-diethylaniline·HCl/4-aminoantipyrine (DEA·HCl/AAP) to produce a chromophore that absorbs at 540 nm. The absorbance due to oxidized DEA·HCl/AAP is directly proportional to the total cholesterol concentration and is measured using a two-filter (540—600 nm) end point technique.

$$\text{Cholesterol Esters} \xrightarrow{\text{CE}} \text{Cholesterol} + \text{Fatty Acids}$$

$$\text{Cholesterol} + O_2 \xrightarrow{\text{CO}} \text{Cholest-4-ene-3-one} + H_2O_2$$

$$2H_2O_2 + \text{DEA·HCl/AAP} \xrightarrow{\text{HPO}} 4H_2O + \text{Oxidized DEA·HCl/AAP}$$

REAGENTS:

Compartment[a]	Form	Ingredient	Quantity[b]	Source
#1	Liquid	CO and HPO	0.25 kU 12 kU	Microbial Plant
#2	Liquid	CE	1.5 kU	Fungal
#3	Tablet	AAP Buffer and Activator	4 μmol	
#4	Liquid	DEA·HCl and Surfactant	8.8 μmol	

a. Compartments are numbered 1-7 with compartment #7 located closest to pack fill position #2.

b. Nominal value at manufacture.

PRECAUTIONS:

USED PACKS CONTAIN HUMAN BODY FLUIDS; HANDLE WITH APPROPRIATE CARE.

FOR *IN VITRO* DIAGNOSTIC USE

MIXING & DILUTION:

The aca automatically aspirates a 0.020-ml sample of serum from the sample cup and injects it into the pack, along with 4.980 ml of Purified Water. The sample cup must contain a sufficient quantity of serum to accommodate the 0.020-ml sample size plus the 0.120-ml "dead volume" of the cup. Precise filling of the cup by the operator is not required. The micro sample cup insert, with a total volume of 0.500 ml and a "dead volume" of 0.010, may also be used.

STORAGE INSTRUCTIONS:

Store under refrigeration (2—8°C). Do not freeze. Do not expose packs to temperatures above 35°C. Do not expose packs to direct sunlight.

EXPIRATION:

Refer to EXPIRATION DATE on the tray label.

SPECIMEN COLLECTION:

Normal procedures for collecting and storing serum may be used for samples to be analyzed by the aca CHOL method.[1]

KNOWN INTERFERING SUBSTANCES[1,2]

- Preliminary test data indicate that heparin does not interfere with this method. These studies indicate that EDTA, oxalate, and citrate may cause a negative interference.[1]
- The following substances and concentrations have been shown to have *no* measurable effect on this method

Ascorbic Acid	3 — 15 mg/dl
Bilirubin	1.6 — 8.5 mg/dl
Calcium	20 mg/dl
Creatinine	2 — 15 mg/dl
Ethanol	25 — 300 mg/dl
Glucose	200 — 500 mg/dl
Hemoglobin	50 — 250 mg/dl
Magnesium	8 — 20 mg/dl
Sodium Bromide	11 mg/dl
Urea	80 — 200 mg/dl
Uric Acid	14 — 35 mg/dl

- Endogenous bilirubin concentrations in excess of 3.5 mg/dl will significantly depress CHOL results.[1]

PROCEDURE:

TEST MATERIALS

Quantity	Item	Du Pont Cat. #
1	aca CHOL Test Pack	702236901
1	Sample System Packet	701989901
	or	
1	Micro Sample System Packet	702694901
	and	
	Micro Sample System Holder	702785000
	Dylux® Photosensitive Printer Paper	700036000
	Purified Water	704209901
	Cell Wash Solution	701868901

®Registered trademark, E. I. du Pont de Nemours & Co., Inc.
Illustration Def.

TEST STEPS

When running analytical test packs, the operator need be concerned only with loading the sample and appropriate test packs into a properly prepared instrument. The aca automatically advances the packs through the test steps and prints the result. For details of sample preparation and pack processing, refer to Section III of the aca Instrument Instruction Manual.

Preset Cholesterol Test Conditions

• Sample Size:	0.020 ml (20 µl)
• Diluent:	Purified Water
• Test Temperature:	37.0 ± 0.1°C
• Reaction Period (Initiation to measurement):	261.5 seconds
• Wavelengths:	540 and 600 nm
• Type of Measurement:	Two filter, end point
• Decimal Point Location	0000 mg/dl
aca I/II:	
• Assigned Starting Point:	0016
• Scale Factor	0.1695 (mg/dl)/count[a]
aca III	
• Assigned Offset C$_0$:	1.600 E1
• Linear Term C$_1$:	−1.761 E0 mg/dl

[a] *The preset scale factor (linear term) was calculated from an absorbance to concentration relationship (sensitivity) of 0.530 mA (mg/dl). Due to small differences in filters and electronic components between instruments, the actual scale factor (linear term) may differ from that given above.*

CALIBRATION

The general calibration procedure is described in the aca Instrument Instruction Manual.

The following information should be considered when calibrating the aca CHOL channel:

• Range of Linearity:	50 — 800 mg/dl
• Reference Materials:	Primary aqueous standards, or secondary calibrators.[a]
• Suggested Calibration Levels	400, 200, and 100 mg/dl[a]
• Starting Point (Offset C$_0$) Adjustment:	For aca I, use the channel #4 adjustable zero offset (ZO) for the last two digits of the starting point. If adjustment of the first two digits of the starting point is required, replace the photo-

	meter method switching board.
	For aca II, use adjustable starting point for all four digits.
	For aca III, enter offset C_o into Method Memory.
• Scale Factor (Linear Term C_1) Adjustment:	May be required for different pack lots.
• Count By (aca I/II):	One (1)
• Readout Units:	The aca prints out in 1-mg/dl increments.

d Primary cholesterol standards prepared in organic solvents such as isopropanol cannot be used for aca calibration because cholesterol will precipitate when these standards are diluted with water in the aca pack. Only primary aqueous cholesterol standards that do not precipitate in the aca pack aqueous dilution step (e.g. Boehringer Mannheim Corporation Precinet* Cholesterol*) should be used for calibration of the aca CHOL method.

*The following instructions are supplementary to the manufacturer's product insert sheet.

Store at room temperature.

If Precinet* standards become turbid upon exposure to cold temperatures, allow them to remain at room temperature for one hour. When standards are clear, invert and follow aca calibration instructions.

If turbid standards do not clear after one hour, discard them.

The calibration procedure using primary cholesterol standards does not check the action of cholesterol esterase. (The esterase reaction has no effect on calibration because cholesterol esterase is not rate limiting.) With each new pack lot, the entire pack chemistry may be checked with an elevate CHOL control as follows:

1. Following calibration of the first CHOL lot with primary standards, establish an aca CHOL bottle value on a control material containing elevated CHOL (300 – 600 mg/dl). Use at least five separate vials and three packs per vial. Calculate the overall mean, the mean for each vial, and the standard deviation of the means.
2. After calibration of subsequent pack lots with primary standards, assay one vial of the above CHOL control material using three packs. The mean should be within the ± 2SD range determined in the previous step.

e Some elevated cholesterol control products contain either artificially added cholesterol esters (e.g. cholesterol acetate) or lipoproteins that are altered during the control product preparation. These materials do not react fully with the enzymatic cholesterol procedures. Therefore, cholesterol bottle values assigned by the manufacturer will not be obtained using the aca CHOL method unless the manufacturer employed a similar enzymatic assay.

f The linearity of CHOL should be verified above the maximum calibration level for each pack lot. The average of triplicate determinations on an elevated patient sample (400 – 800 mg/dl) diluted into the calibration range should be equivalent to the result calculated from the average of triplicate determinations on the undiluted factor serum and the dilution factor.

*Registered trademark, Boehringer Mannheim Corp., New York, N.Y.

QUALITY CONTROL

Two types of quality control procedures are recommended:

- **General Instrument Check.** Refer to the Filter Balance Procedure and the Absorbance Test Method described in the aca Instrument Instruction Manual. Refer also to the ABS Test Methodology literature.
- **Cholesterol Method Check.** At least once daily run a CHOL test pack on a solution of known cholesterol concentration such as an assayed control or calibration standard other than that used to calibrate the CHOL channel. For further details review the Quality Assurance Section of the aca Chemistry Instruction Manual. The result obtained should fall within acceptable limits defined by the day-to-day variability of the system as measured in the user's laboratory. (See SPECIFIC PERFORMANCE CHARACTERISTICS for guidance.) If the result falls outside the laboratory's acceptable limits, follow the procedure outlined in the Chemistry Troubleshooting Section of the aca Chemistry Instruction Manual.

A standard deviation for five consecutive packs greater than 5 mg/dl for a level of 225 mg/dl or greater than 9 mg/dl for a level of 750 mg/dl indicates a possible system malfunction.

RESULTS:

The aca automatically calculates and prints the concentration of CHOL in mg/dl using the general scheme #1 illustrated in the Calculation of Results Section of the aca Chemistry Instruction Manual.

Information specific to the CHOL calculation is listed below:

aca I/II

• Count By:	One (1)
• Scale Factor	0.1695 (mg/dl)/count

aca III

• Linear Term C_1	−1.761 E0 mg/dl

LIMITATION OF PROCEDURE:

aca readouts in excess of 800 mg/dl should be repeated after diluting the sample with Purified Water to produce a sample concentration within the range of linearity. The resulting readout must then be multiplied by the dilution factor to give the CHOL concentration of the undiluted sample.

The aca reporting system contains error messages to warn the operator of specific malfunctions. Any report slip containing a letter code or word immediately following the numerical value should be held for follow-up. Refer to the aca Instrument Instruction Manual.

NORMAL RANGE:

120 — 280 mg/dl (serum)[a, 3]

A more extensive literature study groups the normal ranges by age.[b, 3]

0 — 19 years	120 — 130 mg/dl
20 — 29 years	120 — 240 mg/dl
30 — 39 years	140 — 270 mg/dl
40 — 49 years	150 — 310 mg/dl
50 — 59 years	160 — 330 mg/dl

Each laboratory should establish its own normal ranges for CHOL as performed on the aca.

a. Normal range data on fasting samples from apparently healthy individuals.

101 males (ages 18—60 years) and 85 females (ages 18—60 years).

b. These normal ranges are cited to emphasize the age dependent variation of cholesterol normal values. This normal range study was performed using a Liebermann-Burchard procedure. Based on the excellent correlation between aca and Liebermann-Burchard methods in field evaluation, an aca normal range derived from a larger population is expected to be similar to the literature ranges cited.

SPECIFIC PERFORMANCE CHARACTERISTICS:

PRECISION[c]

Within-Run[d]

MEAN	S.D.	C.V. (%)	N
174.0 mg/dl	2.1	1.2	20
493.6 mg/dl	6.0	1.2	20

Day-to-Day[e]

MEAN	S.D.	C.V. (%)	N
157.6 mg/dl	2.4	1.5	34
266.8 mg/dl	3.4	1.3	34

LINEARITY

50—800 mg/dl

c. All SPECIFIC PERFORMANCE CHARACTERISTICS tests were run after normal recommended equipment quality control checks were performed (see aca Instrument Instruction Manual).

d. N test packs, in series with a sample cup containing human serum, were used.

e. One test pack per day for N days. A fresh sample of lyophilized serum base was used each day.

BIBLIOGRAPHY:

Stadtman, T.C., "Methods in Enzymology, Vol. III," Colowick, S.P. and Kaplan, N.O. (Eds.), Academy Press, New York, N.Y., 1957, pp. 392—394.

Stadtman, T.C., "Methods in Enzymology, Vol. I," Colowick, S.P. and Kaplan, N.O. (Eds.), Academy Press, New York, N.Y., 1955, pp. 678—681.

Flegg, H.M., *Anal. Clin. Biochem.*, 10, p. 79 (1973).

[2] Roschlau, P., Bernt, E., and Gruber, W., *Z. Klin. Chem. Klin. Biochem.*, 12, p. 403 (1974).

[3] Roschlau, P., Bernt, E., and Gruber, W., *Clin. Chem.*, 21, p. 941 (1975).

[4] Richmond, W., *Clin. Chem.*, 19, p. 1350 (1973).

[5] "Simultaneous Cholesterol/Triglycerides," Technicon Corp., Tarrytown, N.Y., publication #AA 11, 24 May, 1971.

[6] Martin, R.F., Mantey, C., Mighore, P.J., Barklow, J.J., Young, J.K., Diorio, A., Buland, A.M. and Huczko, L., "An Evaluation of a Cholesterol Method for the Du Pont aca," January, 1976.[m]

[7] Westgard, J.O., Carey, R.N., and Wold, S., *Clin. Chem.*, 20, p. 825 (1974).

[8] Colove, E., Harris, E.K., and Williams, G., *J. Clin. Chem.*, 16, p. 1028 (1970).

[9] Tietz, N.W., Fundamentals of Clinical Chemistry, W.B. Saunders Co., Philadelphia, Pa., 1970, p. 42.

[10] Supplementary information pertaining to the effects of various drugs and patient conditions on *in vivo* or *in vitro* diagnostic test levels can be found in "Drug Interferences with Clinical Laboratory Tests," *Clin. Chem.*, 21, April, 1975. (This is an update of an earlier compendium — *Clin. Chem.*, 18, October, 1972.)

[11] Rautela, G.S. and Liedtke, R.J., "Automated Enzymic Measurement of Total Cholesterol in Serum," *Clin. Chem.*, 24, (1978).

[12] Fredrickson, D.S., Levy, R.I., and Lees, R.S., *New Engl. J. Med.*, 276, p. 148 (1967).

m. Reprints available from the Du Pont Co., Instrument Products, Automatic Clinical Analysis Division.

aca
DU PONT AUTOMATIC CLINICAL ANALYZER

aca TEST METHODOLOGY

TGL
TRIGLYCERIDE

INTENDED USE:

The TGL pack is used in the Du Pont Automatic Clinical Analyzer (aca) to quantitatively measure triglycerides in serum.

SUMMARY:

The aca triglyceride (TGL) method is the first reported enzymatic technique to combine the use of lipase and glycerol dehydrogenase[1] in a kinetic measurement for serum triglycerides.[2] Fluctuations in the relative concentration of tri-, di- and monoglycerides and free glycerol which occur in patient samples upon storage[3,4] are automatically accommodated since the aca TGL method measures the sum of these metabolites. The contribution of di- and monoglycerides is negligible.[5]

Split sample comparison between the aca method and an automated enzymatic procedure which also measures free glycerol[6] gave a correlation coefficient of 0.995 (correlation slope = 0.886, correlation intercept = 4.5 mg/dl).[7] Compared to a semi-automated Levy-Keyloun procedure,[8] the aca gave a correlation coefficient of 0.972 with an 18.4 mg/dl-bias due in part to the exclusion of endogenous glycerol measurement in the alternate method.[7]

PRINCIPLES OF PROCEDURE:

An enzymatic technique is used in which lipase converts triglycerides to free fatty acids and glycerol. The glycerol thus formed is converted by glycerol dehydrogenase (GDH) to dihydroxacetone with the simultaneous reduction of NAD.

$$\text{Triglycerides} \xrightarrow{\text{Lipase}} \text{Glycerol} + \text{Fatty Acids}$$

$$\text{Glycerol} + \text{NAD}^{+} \xrightarrow{\text{GDH}} \text{Dihydroxacetone} + \text{NADH} (+\text{H}^{+})$$

The change in absorbance at 340 nm due to the formation of NADH over a 17.07-second measurement period is directly proportional to the total amount of glycerol and its precursors (TGL) in the sample.

REAGENTS:

Compartment[a]	Form	Ingredient	Quantity[b]	Source
#1	Liquid	Buffer		
#2	Liquid	Lipase and Stabilizer	6200 IU	Fungal
#3	Tablet[c]	NAD	27 μmol	
#5	Liquid	Glycerol dehydrogenase and Stabilizer	5 IU	Bacterial
#6	Liquid	Surfactant & Stabilizer		
#7	Tablet[c]	Buffer		

a Compartments are numbered 1-7, with compartment #7 located closest to pack fill position #2

b Nominal value at manufacture.

c Tablet contains excipients

PRECAUTIONS:

USED PACKS CONTAIN HUMAN BODY FLUIDS; HANDLE WITH APPROPRIATE CARE.

FOR *IN VITRO* DIAGNOSTIC USE

MIXING & DILUTION:

The aca automatically aspirates a 0.040-ml sample of serum from the sample cup and injects it into the pack, along with 4.960 ml of Purified Water. The sample cup should contain a sufficient quantity of serum to accommodate the 0.040-ml sample size plus the 0.120-ml "dead volume" of the cup. Precise filling of the cup by the operator is not required. The micro sample cup insert, with a total volume of 0.500 ml and a "dead volume" of 0.010 ml, may also be used.

STORAGE INSTRUCTIONS:

Store under refrigeration (2—8°C). Do not freeze. Do not expose sealed trays to temperatures above 35°C. Do not expose packs to direct sunlight.

EXPIRATION:

Refer to EXPIRATION DATE on the tray label

SPECIMEN COLLECTION:

Normal procedures for collecting and storing serum may be used for samples to be analyzed by the aca TGL method.[10]

Blood collection tubes in which stoppers have been lubricated with glycerol should be avoided since they will cause erroneously elevated results.

KNOWN INTERFERING SUBSTANCES[11]

- The following substances and concentrations have been shown to have *no* measurable effect on this method:[12]

Urea	100 mg/dl
Creatinine	50 mg/dl
Bilirubin	25 mg/dl
Hemoglobin	200 mg/dl
Lithium	12.5 mg/dl
Zinc	500 µgm/dl
Lecithin	40 mg/dl
α-Glycerophosphate	2 mg/dl
Glucose	500 mg/dl

- The effect of various anticoagulants has not been evaluated. Restrict use to serum.
- The single wavelength measurement used in this method eliminates interferences from chromophores whose 340-nm absorbance is constant throughout the measurement period.
- Glycerol and other polyols will react in the aca TGL method and may cause positive interference.

PROCEDURE:

TEST MATERIALS

Quantity	Item	Du Pont Cat. #
1	aca TGL Test Pack	702230901
1	Sample System Packet or	701989901
1	Micro Sample System Packet and	702694901
	Micro Sample System Holders	702785000
	Ovion* Photosensitive Printer Paper	700036000
	Purified Water	704209901
	Cell Wash Solution	701864901

*Registered trademark, E. I. du Pont de Nemours & Co., Inc., Wilmington, Del.

TEST STEPS

When running analytical test packs the operator need be concerned only with loading the sample and appropriate test packs into a properly prepared instrument. The aca automatically advances the packs through the test steps and prints the result. For details of sample preparation and pack processing, refer to Section III of the aca Instrument Instruction Manual.

Preset Triglyceride Test Conditions

• Sample Size:	0.040 ml (40 µl)
• Diluent:	Purified Water
• Test Temperature:	37.0 ± 0.1°C
• Reaction Period (Initiation to measurement):	39.5 seconds
• Measurement Period	17.07 seconds
• Wavelength:	340 nm
• Type of Measurement:	Rate
• Decimal Point Location	0000. mg/dl
aca I/II:	
• Assigned Starting Point:	9950.
• Scale Factor:	0.7428 (mg/dl)/count[d]
aca III:	
• Assigned Offset C$_0$:	−5.000 E1 (−5.0 × 10^1)
• Linear Term C$_1$:	1.486 E1 mg/dl[d]

d. The preset scale factor (linear term) was calculated from an absorbance to concentration relationship (sensitivity) of 0.2356 mA/min/(mg/dl). Due to small differences in filters and electronic components between instruments, the actual scale factor (linear term) may differ from that given above.

CALIBRATION

The general calibration procedure is described in the aca Instrument Instruction Manual.

The following information should be considered when calibrating the aca TGL channel:

• Range of Linearity:	0 — 500 mg/dl
• Reference Materials:	Primary standards* or secondary calibrators.†
• Suggested Calibration Levels:	500, 300, 100 mg/dl
• Starting Point (Offset C$_0$) Adjustment:	For aca I, use the channel #9 adjustable zero offset (Z0) for the last two digits of the starting point. If adjust-

ment of the first two digits is required, reface the photometer method switching board.

For aca II, use adjustable starting point for all four digits.

For aca III, enter offset C_o into Method Memory.

- Scale Factor (Linear Term C_1) Adjustment: May be required for different pack lots.
- Count By (aca I/II): Two (2)
- Readout Units: The aca I/II prints out in mg/dl increments; aca III prints out in 1 mg/dl increments.

e. Preparation of Primary Standards

1. Prepare a stock glycerol solution equivalent to 1000 mg/dl TGL by weighing 1.0952 ± 0.0001 g of 96% glycerol into a 50-ml Erlenmeyer flask. The glycerol should be weighed in a low humidity environment and the cap of the bottle replaced immediately after use. It is recommended that analytical grade glycerol be used and be purchased in small bottles and stored in a desiccator. Dilute the glycerol with about 25 ml of Purified Water and transfer the solution to a 1000 ml volumetric flask. To assure complete transfer, the Erlenmeyer should be rinsed with five 25-ml portions of Purified Water. Adjust the final volume to 1000 ml and mix well.

2. If a balance capable of the required precision is not available, a stock glycerol solution equivalent to the above may be prepared as follows:

   Carefully pipet 1.00 ml of glycerol into a 1000 ml volumetric flask using a 1.0-ml "to contain" pipet. Since commercial glycerol solutions are quite viscous, the glycerol will adhere to the internal surface of the pipet. Therefore, the liquid should not be drawn above the mark. We suggest that you select a pipet with a large bore at the tip. Allow the pipet to drain into the volumetric flask for several minutes, then rinse the pipet into the flask with a minimum of 25 ml of Purified Water. Adjust the final volume to 1000 ml (10.00 dl) and mix well.

   The TGL concentration of this stock solution is given by the following equation:

$$\text{mg TGL/dl} = \frac{P \times D \times V_i \times \frac{875}{92} \times 1000}{V}$$

$$= P \times D \times 951.1$$

where:
- P = purity of glycerol (given on label, e.g. 96% purity = to $P = 0.96$)
- D = density of glycerol (g/ml) = specific gravity given on label
- V_i = 1.00 ml = volume of glycerol added to volumetric flask
- 875 = average molecular weight of serum triglycerides[1]
- 92 = molecular weight of glycerol
- 1000 = factor to convert g to mg
- V = 10.00 dl = total volume of stock solution

3. Make appropriate dilutions of the stock standard using Purified Water.
4. Stock and working standards are stable in closed containers for at least six months at 4°C.
5. The calibration procedure using glycerol standards does not check the activity of lipase. (The lipase reaction has no effect on calibration but does affect sample readings.) With each new pack lot, the entire pack chemistry may be checked with an elevated TGL control as follows:
   (a) Following calibration of the first TGL lot with glycerol standards, establish an aca TGL bottle value on a control material[f] containing elevated TGL (350—450 mg/dl). Use at least five separate vials and three packs per vial. Calculate the overall mean, the mean for each vial, and the standard deviation of the means.
   (b) After calibration of subsequent pack lots with glycerol standards, assay one vial of the above TGL control material using three packs. The mean should be within the ±2 SD range determined in the previous step.

f. Most commercial control products contain significant quantities of glycerol. Therefore, triglyceride bottle values assigned by the manufacturer will not be obtained using the aca TGL method unless the manufacturer's method of value assignment measures glycerol as well as triglycerides.

QUALITY CONTROL

Two types of quality control procedures are recommended.

- **General Instrument Check.** Refer to the Filter Balance Procedure and the Absorbance Test Method described in the aca Instrument Instruction Manual. Refer also to the ABS Test Methodology literature.
- **Triglyceride Method Check.** At least once daily run a TGL test pack on a solution of known TGL concentration such as a properly assayed control[f] or calibration standard other than that used to calibrate the TGL channel. For further details review the Quality Assurance Section of the aca Chemistry Instruction Manual. The result obtained should fall within acceptable limits defined by the day-to-day variability of the system as measured in the user's laboratory. (See SPECIFIC PERFORMANCE CHARACTERISTICS for guidance.) If the result falls outside the laboratory's acceptable limits, follow the procedure outlined in the Chemistry Troubleshooting Section of the aca Chemistry Instruction Manual.

A standard deviation for five consecutive packs greater than 9 mg/dl for a level of 100 mg/dl or greater than 15 mg/dl for a level of 500 mg/dl indicates a possible system malfunction.

RESULTS

The aca automatically calculates and prints the concentration of TGL in mg/dl using the general scheme #2 illustrated in the Calculation of Results Section of the aca Chemistry Instruction Manual.

Information specific to the TGL calculation is listed below:

aca I/II:

- Count By: Two (2)
- Scale Factor: 0.7428 (mg/dl)/count[1]

aca III

- Linear Term C_1: 1.486 E1 mg/dl[m]

LIMITATION OF PROCEDURE:

aca readouts in excess of 500 mg/dl should be repeated after diluting the sample with Purified Water to produce a sample concentration within the range of linearity. The resulting readout must then be multiplied by the dilution factor to give the TGL concentration of the undiluted sample.

The aca reporting system contains error messages to warn the operator of specific malfunctions. Any report slip containing a letter code or word immediately following the numerical value should be held for follow-up. Refer to the aca Instrument Instruction Manual.

NORMAL RANGE:

30 — 200 mg/dl[7,9]

A more extensive study from the literature groups the normal ranges by age.[7,n]

0 — 29 years	20 — 150 mg/dl
30 — 39 years	20 — 160 mg/dl
40 — 49 years	20 — 170 mg/dl
50 — 59 years	20 — 200 mg/dl

g. Normal range data on fasting samples from apparently healthy individuals:

171 males (ages 9—65 years) and 76 females (ages 5—64 years).

n. The literature normal ranges cited have been adjusted 10 mg/dl upward to accommodate the average serum free glycerol content.[7,10,11] Each laboratory should establish its own normal ranges for TGL as performed on the aca.

SPECIFIC PERFORMANCE CHARACTERISTICS:

PRECISION[i]

Within-Run[j]

MEAN	S. D.	C. V. (%)	N
131 mg/dl	5	3.8	20
396 mg/dl	9	2.3	20

Day-to-Day[k]

MEAN	S. D.	C. V. (%)	N
132 mg/dl	8	6.1	30
388 mg/dl	9	2.3	24

LINEARITY

0 — 500 mg/dl

i. All SPECIFIC PERFORMANCE CHARACTERISTICS tests were run after normal recommended equipment quality control checks were performed (see aca Instrument Instruction Manual).

j. N test packs, in series with a sample cup containing lyophilized serum base material, were used.

k. One test pack per day for N days. A fresh sample of lyophilized serum base material was used each day.

BIBLIOGRAPHY:

[1] Hagen, J. H., and Hagen, P. B.: *Can. J. of Biochem. and Physiol.* **40**, p. 1129 (1962).

[2] Rautela, G. S., Hall, R. G., Jr., Bekiesz, C. L., and Wermus, G. R.: *Clin. Chem.* **20**, p. 847 (1974). Presented at the 26th National Meeting of the American Association of Clinical Chemists (AACC), August, 1974.

[3] Tiffany, T. O., Morton, J. M., Hall, E. M., Garrett, A. S., Jr.: *Clin. Chem.* **20**, p. 476 (1974).

[4] Rautela, G. S., Slater, S., and Arvan, D. A.: *Clin. Chem.* **19**, p. 1193 (1973).

[5] Hirsch, J., and Ahrens, E. J., Jr.: *J. Biol. Chem.* **233**, p. 311 (1958).

[6] Buccolo, G., and David, H.: *Clin. Chem.* **19**, p. 476 (1973).

Westgard, J. O., Carey, R. N., and Gadsden, R. H.: "An Evaluation of a Triglyceride Method for the Du Pont aca," August, 1975.[m]

[7] Levy, A. L., and Kevloun, C.: "Advances in Automated Analysis," Technicon International Congress, Vol. 1, p. 497, 1970.

[8] Westgard, J. O., University of Wisconsin Medical Center, Madison, Wisc., June, 1974. (Private communication.)

[9] Tietz, N. W.: "Fundamentals of Clinical Chemistry," W. B. Saunders Co., Philadelphia, Pa., 1970. (General specimen collection: p. 42; average molecular weight of serum triglycerides: p. 338.)

[10] Supplementary information pertaining to the effects of various drugs and patient conditions on *in vivo* or *in vitro* diagnostic levels can be found in "Drug Interferences with Clinical Laboratory Tests," *Clin. Chem.*, 21, April, 1975. (This is an update of an earlier compendium — *Clin. Chem.* **18**, October, 1972.)

[11] The Du Pont Co., Instrument Products, Automated Clinical Analysis Division. (Unpublished report.)

[12] Fredrickson, D. S., Levy, R. J., and Lees, R. S.: *New England Journal of Medicine*, **276**, pp. 34-44 (1967).

[13] Mourad, N., Zager, R., and Neveu, P.: *Clin. Chem.*, **19**, p. 116 (1973).

[14] Olson, A. O., and Brennan, R. L.: *Clin. Chem.*, **19**, p. 1060 (1973).

m. Reprints available from the Du Pont Co., Instrument Products, Automatic Clinical Analysis Division.

APPENDIX G

FISHER-FAIRBANKS WALKING TEST

1875 • Brigham Young University Centennial • 1975
College of Physical Education

October 15, 1979

Susan Quaal
569 10th Avenue
Salt Lake City, UT 84103

Dear Susan,

Enclosed are two pages that describe the method for giving the Fisher-Fairbanks walking test. If you need to reference this test use the following reference:

J. George Fairbanks, A Submaximal Walking Test: Prediction of Max VO_2 and Physical Fitness in Adult Males. A Dissertation, August 1978.

Sincerely,

Garth Fisher, Ph.D., Director
Human Performance Research Center

Human Performance Research Center, 116 Richards Building, Brigham Young University, Provo, Utah 84602
(801) 374-1211, Extension 3981

1. Measure exactly 100 feet on the testing area and clearly mark this distance so that the rate of the walk can be measured accurately.

2. The subject should rest for at least 10 minutes and then measure the number of radial pulse beats in one minute.

3. If the test is self-administered, carry the stopwatch with you as you take the walking test. Begin the test by walking in a normal walking style but walk briskly as if you are late to class or late to work.

4. Measure the time it takes to cover 100 feet; adjust the walking pace so that the rate for 100 feet is between 15 and 16 seconds (4.26-4.55 mph or 6.82-7.28 km/hr). Monitor the pace periodically, for consistency.

5. After walking for 5 minutes at a consistent pace, come to a standstill and take your radial pulse rate immediately by starting stopwatch on the first recovery heart beat (designate this beat as zero) and by counting the number of heart beats in exactly 10 seconds. If you fail to find the radial pulse within two seconds following the completion of the wald, you must start the walking test over again with step 3.

6. Max. VO_2 can be estimated by placing the appropriate figures in the following regression equation (equation 10, Table 4):

Max. VO_2 (ml/kg.min) = 111.6 - 0.06198 (WT) - [0.4564 (RecHR)] - 0.0867 (Rest HR)

where weight is in pounds, recovery heart rate from 0 to 10 seconds is in beats per minute, and resting heart rate is in beats per

minute. The standard error of estimation of this regression equation is $\pm$ 5.74 ml/kg.min and the percent error in predicting the mean max. VO_2 value is 11.9%.

7. Physical fitness levels can be categorized according to the following norms:

Max. VO_2	Physical Fitness Category
below 29 ml/kg.min	Very Poor
30-39 ml/kg.min	Poor
40-49 ml/kg.min	Average
50-59 ml/kg.min	Good
above 60 ml/kg.min	Excellent

These norms were developed from a histogram of the Max. VO_2 data collected in this study. They are similar to the norms presented by Cooper (1970) and Sharkey (1974).

APPENDIX H

COVER LETTER ACCOMPANYING COPY OF LIPID PROFILE SENT TO EACH SUBJECT

Ms. Susan J. Quaal, RN CVS BSN
569 10th Avenue
Salt Lake City, Utah 84103
December 29, 1979

Dear Mr. _____

Enclosed, please find a copy of your laboratory lipid results that were drawn during your cardiovascular screening at the YMCA on December 18, 19 or 20. The two values which you should be concerned with are the total cholesterol (Abbreviated TOT CHOL) and triglycerides (Abbreviated TRIGLY). The LDL CHOL, HDL CHOL, and VLDL CHOL values are fractioned breakdown values of your total cholesterol which are of interest to my research investigation only.

Comments regarding your lipid profile:

Cholesterol:

Triglycerides:

As you requested on your cardiovascular screening questionnaire, I (did did not) send a copy of this report to your personal physical, Dr. _____.

Also, forthcoming, later in January, will be the results of your fitness tests. The pre and postexercise pulses recorded need to be computed in a regression-equation to compute your oxygen uptake--I can't acquire computer time for this calculation until later in the month. The oxygen uptake will enable us to categorize your overall cardiovascular fitness as poor, fair, good, or excellent, and I'm sure this information would also be of interest to you.

Again, thank you for your participation and cooperation. If you have any additional questions, please feel free to contact me at 328-0011.

Happy New Year!

Sincerely,

Susan J. Quaal, RN CVS BSN

REFERENCES

Abbey, J. C. Concepts and principles. In J. Smith (Ed.), Five years to improve curricula in western schools of nursing. Boulder, Colorado: Western Interstate Commission for Higher Education, 1972.

Abere, J. J. Quantitation of apoliproprotein A-1 of human high density lipoprotein. Metabolism, 1975, 25, 411.

American Heart Association. Abstracts of the 33rd annual meeting--Council on Arteriosclerosis, 1979, 1-20.

Åstrand, I.; Åstrand P.; & Rodahl, K. Maximal heart rate during work in older men. Journal of Applied Physiology, 1959, 14, 562.

Åstrand, I. Aerobic work capacity in men and women with special references to age. Acta Physiology Scandinavia, 1960, 49. (Supplement 169)

Åstrand, P-O. & Rodahl, K. Textbook of work physiology-physiological bases of exercise. New York: McGraw-Hill, 1977.

Bang, H. D. Plasma lipid and lipoprotein pattern in Greenlandic west-coast Eskimos. Lancet, 1971, 1, 1143.

Behnke, A. R. & Wilmore, J. H. Evaluation and regulation of body build. Englewood Cliffs, New York: Prentice-Hall, 1974.

Bethell, H. B. N. & Nixon, P. G. F. Understanding the atrial sound. British Heart Journal, 1973, 35, 229-235.

Blumenthal, J. A. Coronary-prone behavior and angiographically documented coronary disease. Paper presented at the Annual Meeting of the American Psychosomatic Society, New Orleans, Louisiana, March 21, 1975.

Brink, P. & Wood, M. Basic steps in planning nursing research. Belmont, California: Wadsworth Publishing Co., 1978.

Bobbert, A. C. Energy expenditure in level and grade walking. Journal of Applied Physiology, 1960, 15, 1015-1121.

Brooks, G. W. & Mueller, E. F. Serum urate concentration among university professors. Journal of American Medical Association, 1966, 195, 415-418.

Burt, D. O. Some chemical factors in the pathogenesis of atherosclerosis. Circulation, 1953, 8, 651-643.

Campbell, D. E. Influence of several physical activities on serum cholesterol concentrations in young men. Journal of Lipid Research, 1965, 6, 478-480.

Campbell, D. E. Influence of diet and physical activity on blood serum cholesterol of young men. American Journal of Clinical Nutrition, February 1966, 18, 79-85.

Campbell, D. E. & Lumsden, T. B. Serum cholesterol concentrations during physical training and during subsequent detraining. American Journal of Medical Science, February 1967, 253, 155-162.

Campbell, D. E. Effect of controlled running on serum cholesterol of young adult males of varying morphological constitutions. Research Quarterly, March 1968, 39, 47-53.

Carlson, I. A. & Earicsson, M. Quantitative and qualitative serum lipoprotein analyses. Part 2. Studies in male survivors of myocardial infarction. Atherosclerosis, 1975, 21, 435-437.

Casselli, W. P. Alcohol and blood lipids: The cooperative lipoprotein phenotyping study. Lancet, 1977, 2, 153-159.

Cooper, K. H. Quantifying physical activity--how and why. Proceedings of the national workshop on exercise in the prevention, in the evaluation, and in the treatment of heart disease. Supplement to the Journal of the South Carolina Medical Association, December 1969.

Cooper, K. H. Aerobics. New York: M. Evans and Co., 1968.

Dorland's illustrated medical dictionary. Philadelphia: W. B. Saunders Company, 1965.

Dyerberg, J. Eicosapentanoic acid and prevention of thrombosis and atherosclerosis. Lancet, 1978, 2, 117.

Eliot, R. S. (Ed.). Stress and the heart. Mount Kirco, New York: Futuri Publishing Co., 1974.

Fairbanks, J. G. A submaximal walking test: Prediction of maximum VO_2 and physical fitness in adult males. Unpublished doctoral dissertation, Brigham Young University, 1978.

Fisher, G. Personal communication, April 14, 1980.

Folkow, B. & Rubenstein, E. H. Cardiovascular effects of acute and chronic stimulation of the hypothalamic defense area in the rat. Acta Physiologica Scandinavia, 1966, 68, 48-57.

Fox, S. M., III. Physical activity and coronary heart disease. American Journal of Cardiology, February 1969, 23, 298-303.

Friedman, M. & Roseman, R. H. Association of specific overt behavior pattern with blood and cardiovascular findings. Journal of American Medical Association, 1959, 169, 1286.

Friedman, M. & Roseman, R. H. Changes in the serum cholesterol and blood clotting time in men subject to cyclic variation of occupation stress. Circulation, 1958, 17, 852-861.

Friedman, M.; Rosenman, R. H.; & Byers, S. O. Serum lipids and conjunctival circulation after fat ingestion in men exhibiting type A behavior pattern. Circulation, 1964, 29, 874-886.

Friedman, M. & Rosenman, R. H. Type A behavior pattern: Its association with coronary heart disease. Annals of Clinical Research, 1971, 3, 300-312.

Fyfe, T. Seasonal variation in serum lipids, and incidence and mortality of ischaemic heart disease. Journal of Atherosclerotic Research, May-June 1968, 8, 591-596.

Garrett, H. L. The effects of a strenuous physical conditioning program on coronary risk factors in men. Doctoral dissertation, George Peabody College, 1965.

Glomset, J. A. & Norum, K. R. The metabolic role of leicithincholesterol acyltransferase: Perspectives from pathology. Advanced Lipid Research, 1973, 11, 1-4.

Flueck, G. J. Familial hyper-alpha-lipoprateinemia: Studies in eighteen kindreds. Metabolism, 1975, 24, 1243-1249.

Fredrickson, D. S. It's time to be practical. Circulation, 1975, 51, 209.

Gordon, T. High density lipoprotein as a protective factor against coronary heart disease: The Framingham study. American Journal of Medicine, 1977, 62, 707-712.

Graham, D. T. Psychosomatic medicine. In N. S. Greenfield & R. A. Sternbach (Eds.), Handbook of psychophysiology. New York: Holt, Rinehart & Winston, 1972.

Gsell, D. & Mayer, J. Low blood cholesterol associated with high carolic, high saturated fat intakes in a Swiss alpine village population. American Journal of Clinical Nutrition, 1962, 10, 471.

Gutmann, M. C. & Benson, H. Interaction of environmental factors and systematic arterial blood pressure: A review. Medicine, 1971, 50, 543-553.

Guyton, A. C. Textbook of medical physiology (5th ed.). Philadelphia: W. B. Saunders Company, 1976.

Harting, H.; Foreyr, J.; Mitchell, R.; Vlasek, I.; & Gotto, A. Relation of diet to high-density lipoprotein cholesterol in middle-aged marathon runners, joggers and inactive men. The New England Journal of Medicine, 1980, 302, 357-361.

Havel, R. J. High density lipoproteins, cholesterol transport, and coronary heart disease. Circulation, 1979, 60, 1.

Hilton, S. M. Hypothalmic regulation of the cardiovascular system. British Medical Bulletin, 1966, 22, 243-248.

Hokanson, J. E. Effects of displaced aggression on systolic blood pressure. Journal of Abnormal and Social Psychology, 1963, 67, 214-218.

Holloszy, J. O. Effects of a six month program of endurance exercise on the serum lipids of middle-aged men. American Journal of Cardiology, December 1964, 14, 753-760.

House, J. S. Occupational stress as a precursor to coronary disease. In W. D. Gentry (Ed.), Psychological aspects of myocardial infarction and coronary care. St. Louis: C. V. Mosby Co., 1975.

Jacobs, D. Personal communication. April 15, 1980.

Jenkins, P. J. Severity of coronary atherosclerosis related to lipoprotein concentration. British Medical Journal, 1978, 2, 388-392.

Krupp, M. A. & Chatton, M. J. Current medical diagnosis and treatment. Los Altos: Lange Medical Publications, 1978.

Levy, R. I. High density lipoproteins 1978--an overview. Lipids, December 1978, 13(12), 911-913.

MacDonald, I. Statistical studies of recorded energy expenditure of man. Nutritional Abstracts and Reviews, 1961, 31, 261.

MacKenzie, Sir James. Angina pectoris. London: Oxford Medical Publications, 1923.

Mann, G. V. & White, H. S. The influence of stress on plasma cholesterol levels. Metabolism, 1953, 2, 47-58.

Marascuilo. L. A. Statistical methods for behavioral science research. New York: McGraw Hill, 1971.

Martin, R. P.; Haskell, W. L.; & Wood, P. D. Blood chemistry and lipid profiles of elite distance runners. Annals of New York Academy of Sciences, 1977, 301, 346-360.

McGinn, N. F. Psychological correlates of blood pressure. Psychological Bulletin, 1964, 61, 209-219.

Miller, N. E. High density lipoproteins and coronary heart disease: A prospective case-control study. Lancet, 1977, 1, 965-974.

Montoye, H. J. The effects of exercise on blood cholesterol in middle-aged men. American Journal of Clinical Nutrition, March-April 1959, 7, 139-145.

Montoye, H. J.; Epstein, F. H.; & Khelsberg, M. O. Relationship between serum cholesterol and body fatness. American Journal of Clinical Nutrition, June 1966, 18, 397-406.

Morris, J. N. Evidence for the benefits of exercise from epidemiological studies. British Journal of Sports Medicine, 1979, 12, 220-222.

Mjos, O. D. & Thelle, D. S. Family study of high density lipoprotein cholesterol and the relationship to age and sex. Acta Medical Scandinavia, 1977, 201, 323-329.

Narbols, V. Human serum lipoproteins and their interrelationships. Advanced Biological Medical Phepics, 1967, 11, 109-112.

Nichols, A. V. & Smith, L. Effect of very low-density lipoprotein on lipid transfer in serum. Journal of Lipid Research, 1965, 6, 206-212.

Nixon, P. F. G. The human function curve. The Practitioner, 1971, 217, 765-770; 935-945.

Osler, W. The lumieian lectures on angina pectoris. Lancet, 1910, 1, 697-702; 839-844.

Paffenbarger, R. S. (unpublished) Observations on Harvard graduates. The relationship between continuing physical activity and coronary disease as cited in Morris, J. N. Evidence for benefit of exercise from epidemiological studies. British Journal of Sports Medicine, 1979, 12, 220-227.

Paffenbarger, R. S. & Laughlin, M. E. Work activity of longshoremen as related to death from coronary heart disease and stroke. New England Journal of Medicine, 1970, 282, 1109-1114.

Paffenbarger, R. S. & Hale, W. E. Work activity and coronary heart mortality. The New England Journal of Medicine, March 1975, 11, 545-549.

Passmore, P. & Dumin, J. V. G. A. Human energy expenditure. Physiological Review, 1955, 35, 801.

Pollock, M. L. Effects of frequency of training on working capacity, body composition, and circulo-respiratory measures. Doctoral dissertation, University of Illinois, 1967.

Pollock, M. L. The quantification of endurance training programs. In J. H. Wilimore (ed.), Exercise and sport sciences review. New York: Academic Press, 1973.

Pollock, J. L.; Wilmore, J. H.; & Fuchs, S. M. III. Health and fitness through physical activity. New York: John Wiley and Sons, 1978.

Raab, W. Preventative myocardiology: Fundamentals and targets. Springfield: Charles C. Thomas, 1970.

Rahe, R. H. & Arthur, R. J. Life-change patterns surrounding illness experience. Journal of Psychosomatic Research, 1968, 11, 341-350.

Reiff, G. G.; Montage, J. H.; & Remington, R. D. Assessment of physical activity. In M. J. Karnonen & A. J. Barry (Eds.), Physical activity and the heart. Springfield, Illinois: Thomas Company, 1967.

Rosenman, R. H. A predictive study of coronary heart disease. Journal of American Medical Association, 1964, 189, 15-22.

Rosenman, R. H. & Friedman, M. Coronary heart disease in the western collaborative group study: A follow-up experience of four and one-half years. Journal of Chronic Diseases, 1970, 23, 173-190.

Richardson, B. J. The nature, the cause, and the treatment of angina pectoris. New York Medical Journal, 1896, 63, 292-294.

Russek, H. I. Role of heredity, diet and emotional stress in coronary heart disease. Journal of American Medical Association, 1959, 171, 503-508.

Russek, H. I. Role of irrational stress in the etiology of clinical coronary heart disease. Diseases of the Chest, 1967, 52, 1-9.

Schrott, H. Increased coronary mortality in relatives of hypercholesterolemic school children: The muscatine study. Circulation, 1979, 59, 320-326.

Schwane, J. & Cundiff, D. Relationships among cardiorespiratory fitness, regular physical activity and plasma lipids in young adults. Metabolism, July 1979, 28, 771-776.

Seyle, H. The general adaptation syndrome and the diseases of adaptation. Journal of Clinical Endocrinology, 1946, 6, 117-230.

Shane, S. R. A comparison of varying levels of physical condition with serum cholesterol concentrations. American Journal of Cardiology, 1966, 18, 540-547.

Stanford heart disease prevention program: Blood chemistry and lipid profiles of elite distance runners. Annals of New York Academy of Science, 1976, 346-360.

Starling, E. H. The linacre lecture on the law of the heart. London: Longmans, Green and Company, 1918.

Stuld, S. C. The relationship of nutrient intake and exercise to serum cholesterol levels in white males in Evans County, Georgia. American Journal of Clinical Nutrition, 1965, 16, 238-242.

Taylor, H. L.; Jacobs, D.; Schucher, B.; Knudsen, J.; Arther, L.; & Debachest, G. A questionnaire for the assessment of leisure time physical activities. Journal of Chronic Disease, 1978, 31, 741-755.

Taylor, H. L.; Buskirk, E.; & Henschel, A. Maximum oxygen intake as an objective measure of cardiorespiratory performance. Journal of Applied Physiology, 1955, 8, 73.

The national diet--heart study final report. Circulation, 1968, 1, 1-428. (Supplement)

Turpeinen, O. Effect of cholesterol-lowering diet on mortality from coronary heart disease and other causes. Circulation, 1979, 59, 1-7.

VanderWait, W. H. & Wyndham, C. H. An equation for the prediction of energy expenditure of walking and running. Journal of Applied Physiology, 1973, 34, 559.

West, R. O. & Hayes, O. B. Diet and serum cholesterol levels: A comparison between vegetarians and nonvegetarians in a Seventh-day Adventist group. American Journal of Clinical Nutrition, 1968, 21, 835-862.

Wolf, S. Changes in serum lipids in relation to emotional stress during rigid control of diet and exercise. Circulation, 1962, 26, 379-387.

Wolff, H. G. Life stress and body disease: A formation in life stress and body disease. Baltimore: Williams and Wilkins, 1950.

Wood, W. & Haskell, W. L. The effect of exercise on plasma high density lipoproteins. Lipids, 1979, 14, 417-427.

VITA

Name	Susan J. Quaal
Birthdate	July 17, 1949
Birthplace	Breckenridge, Minnesota
Universities	North Dakota State University Fargo, North Dakota
	Concordia College Moorhead, Minnesota
	Minnesota State University Moorhead, Minnesota
	Brigham Young University Provo, Utah
Degrees	
1978	B.S., Brigham Young University Provo, Utah
1977	C.V.S., Arizona Heart Institute Phoenix, Arizona
1969	Diploma, St. Luke's Hospital School of Nursing Fargo, North Dakota
Memberships	American Association of Critical Care Nurses Greater Salt Lake Chapter, AACN American Heart Association American College of Sports Medicine American Heart Association Council on Cardiovascular Nursing
Awards	
1979	Deseret Foundation Grant for investigation of lipid levels in 50 male subjects
1979	The Third International Conference on Human Functioning, Best Student Paper, Wichita, Kansas

Awards (continued) 1979-1980	Who's Who Amongst American Students and Universities
Professional Positions	Staff Nurse, Thoracic ICU, LDS Hospital, Salt Lake City, Utah, September 1977 to present.
	Nurse Specialist Resident (University of Utah affiliation), Cottonwood Hospital, Murray, Utah, 1979.
	Staff Nurse - Charge Nurse, Critical Care In-service Instructor, St. Luke's Hospital, Fargo, North Dakota, 1971-1976.
	Campus Health Nurse, Concordia College, Moorhead, Minnesota, 1970-1971.
	Staff Nurse, St. Luke's Hospital, Fargo, North Dakota, 1969-1970.
Professional Activities	Speaker, American College of Cardiology, Rogers Heart Foundation, 5th Annual Seminar on Cardiovascular Nursing, Miami, Florida, May 1980.
	Speaker, Greater Salt Lake Chapter, AACN, Cardiovascular Nursing Symposium, Park City, Utah, April 1980.
	Speaker, Intermountain Health Care, Nursing Update 1980, Salt Lake City, Utah, March 1980.
	Lecturer, Brigham Young University, College of Nursing, Provo, Utah, 1977 to present.
	Lecturer, University of Utah, Division of Continuing Education, Salt Lake City, Utah, 1977 to present.
	Author, Policy/Procedure Book, Holy Cross Hospital, Salt Lake City, Utah, 1979.

Professional Activities (continued)	Nurse Coordinator, "Heart Club," Utah Heart Association, 1979-1980.
	Program Chairman, Red River Valley Chapter AACN, Fargo, North Dakota, 1973-1976.
	Volunteer Health Nurse, Migrant Health Services, Moorhead, Minnesota, 1974-1975.